December 19, 2003

The Dow Chemical Company
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ECOLOGICAL RISK ASSESSMENT REVIEW

Dear George,

We have reviewed the *Tittabawassee River Aquatic Ecological Risk Assessment* (ERA) conducted by Galbraith Environmental Science LLC (GES), on behalf of the Remediation and Redevelopment Division of MDEQ, and have attached a detailed technical evaluation of the final report. Based on its evaluation Dow believes:

- The GES report can only be considered a screening level ERA, when compared to US EPA Guidelines for conducting an ERA;
- The GES ERA contains numerous errors and technical flaws; and
- Dow will conduct the more definitive, or Tier II, ERA following EPA Guidelines as part of the Remedial Investigation required under the License.

Dow will incorporate all the data collected by MDEQ for the GES ERA in Dow's ERA, conducted under the License.

If you or your staff have any questions on this review, please direct them to John Phillips at (989) 636-1762.

Sincerely,

Susan Carrington
Vice President and Director
Michigan Dioxin Initiative

Attachment

THE DOW CHEMICAL COMPANY'S REVIEW OF THE

TITTABAWASSEE RIVER AQUATIC ECOLOGICAL RISK ASSESSMENT: POLYCHLORINATED DIBENZO-P-DIOXINS, POLYCHLORINATED DIBENZOFURANS

Conducted by Galbraith Environmental Services, LLC

Submitted December 19, 2003

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INTRODUCTION TO THE RESPONSE TO THE GALBRAITH ENVIRONMENTAL SERVICES AQUATIC ECOLOGICAL RISK ASSESSMENT

This document represents conclusions of The Dow Chemical Company (Dow) based on its evaluation of the technical methods, assumptions, and conclusions of the limited aquatic ecological risk assessment (ERA) prepared by Dr. Hector Galbraith of Galbraith Environmental Services LLC (GES) for the Remediation & Redevelopment Division (RRD) of the Michigan Department of Environmental Quality (MDEQ). The GES report is entitled: "*Tittabawassee River Aquatic Ecological Risk Assessment: Polychlorinated Dibenzo-p-dioxins, Polychlorinated Dibenzofurans*" (Report).

EXECUTIVE SUMMARY

Dow agrees with GES that the EPA Guidelines for conducting an ERA should be followed. Yet, when the GES ERA is compared against EPA ERA Guidelines, the GES ERA only meets the threshold for a screening level or Tier I assessment. In addition, Dow has identified numerous errors and flaws in scientific logic, which are outlined in this report. For both these reasons, the GES ERA can not be used for remedial decision making. Dow will conduct a definitive ERA as part of the Remedial Investigation within the terms of its Hazardous Waste Operating Licenses and follow EPA Guidelines.

1. <u>Under EPA Guidelines for Conducting an ERA, the GES Report Can Only Be Considered A Screening Level ERA</u>

Dow agrees that the EPA Guidelines apply to the conduct of an ecological risk assessment. Under the EPA Guidelines the GES ERA only meets the requirements for a screening level or Tier 1 assessment.

- Under EPA Guidelines a screening level assessment deliberately overestimates risks, to avoid excluding a site from further evaluation, when further evaluation may be appropriate. As such, a screening level ERA under EPA Guidelines may use calculated estimates of exposure rather than measured values. Similarly, single values may be used to model toxicity rather than a range of measured values, as was the case in the GES ERA. In the GES ERA, the use of these two factors alone result in exaggerated overestimates of risk in the order of 10 to 100 times.

- The sole use of NOAELs, as used in the GES Report, is most appropriate for Tier I or screening-level assessments. As noted in EPA guidance (EPA, 1997): "*screening ecotoxicity values should represent a NOAEL for long-term (chronic) exposures to a contaminant… A NOAEL is more appropriate than a LOAEL to use as an screening ecotoxicity value to ensure that risk is not underestimated*".

- The following are some examples of the limitations of the GES ERA that restrict its classification to screening-level or Tier I assessment under EPA Guidelines are:
  - Conclusions are based almost exclusively on estimated results (i.e. modeled or extrapolated) rather than measured values collected from the Tittabawassee River area, which leads to significant (10 to 100 times) overestimates of exposure and risk.

- There was an exclusion of site-specific data (measured values) in the Report, for example:
  - ♦ A weight-of-evidence approach in evaluating risks, as prescribed by EPA Guidelines, (1997, 1998) was absent in the Report.
  - ♦ GES excluded walleye sample results without explanation. This fish data showed lower levels of PCDD/PCDF exposure and if used, would have led to lower levels of risk to higher level wildlife, such as pisivorous (fish eating) birds and mammals.
  - ♦ Certain terrestrial animals (e.g., small mammals, earthworms) intended to be considered as biota inputs of PCDDs/PCDFs were excluded due to an inability to collect samples.
  - ♦ GES used deterministic single data-point approaches (i.e. simple math) rather than probabilistic risk assessment methods, which implies a higher level of accuracy and confidence than actually exists.
- The GES ERA relies on single point no-observed adverse effect levels (NOAELs) to estimate toxicity (TRVs) rather than a more complete and robust set of measured values. This practice is consistent with EPA guidance for screening-level assessments (EPA, 1997), yet is not acceptable for a definitive ERA or for remedial decision making.
- An incomplete review of the scientific literature was conducted by GES (which is typical of a screening level ERA), particularly with respect to the toxicity of PCDDs/PCDFs to piscivorous (fish eating) birds, such as bald eagle, great blue herons, and osprey, resulting in errors and misclassification of exposure and risk.
- The GES Report is based on a single exposure route (i.e. eating fish).
- The small number and type of samples (i.e. only 85 fish[1] and 9 duck eggs[2]) taken from the Tittabawassee River area that form the basis of the GES ERA are consistent with a screening level ERA, particularly when compared to more definitive ERAs[3] conducted at other large sites in the U.S. (see Table 1) that provided a higher level of confidence in remedial decision making.

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[1] A total of 94 fish, comprising 5 fish species, were caught and analyzed by MDEQ in 2002, but data from 9 walleye caught and analyzed are not referenced in the GES Report nor does the GES Report indicate why the walleye data were not used in either the piscivorous bird or mink/otter risk analysis.

[2] Data was referenced from 9 wood duck and merganser eggs that were also included in the GES Report, collected by MDEQ.

[3] More definitive ERAs provide increased confidence in remedial decision making by using multiple lines of evidence from wildlife populations collected in site field studies. An example of the use of multiple lines of evidence is the ERA that was conducted at Clinch River in which five lines of evidence were developed for fish and two for piscivorous wildlife (EPA report available at: http://yosemite.epa.gov/water/owrccatalog.nsf). Other examples in which multiple lines of evidence were utilized include the Clark Fork (EPA report available at: http://www.epa.gov/region8/superfund/sites/mt/millterap.html), and Hudson Rivers (EPA report available at: http://www.epa.gov/hudson/revisedbera-plates.pdf). See Table 1.

- The use of estimated, exaggerated values as a basis of risk conclusions indicate the screening-level nature of the GES ERA report. Some of the field measured values used were in direct conflict with conclusions made in the GES Report, for example:
  - Wood duck eggs taken from a control location where no exposure was reported to have occurred exhibited higher concentrations of PCDDs/PCDFs than eggs taken from the Shiawassee Refuge, where exposure was asserted to occur.
  - Despite a large difference between the estimated TEQ levels in eggs and measured TEQ levels in eggs, GES used the higher estimated levels rather than lower measured levels to calculate exaggerated hazard indices.

2. <u>Numerous Technical Flaws Were Found in the GES ERA</u>

In addition to the screening level, Tier-1 nature of the GES ERA, a number of technical flaws exist in the GES Report which compromise, and even invalidate some of its preliminary conclusions. These errors and flaws resulted in over-estimated calculations of risk of PCDDs/PCDFs to piscivorous (fish eating) birds and mammals on the Tittabawassee River by 10 to 100-fold.

For example:

- The report arbitrarily and inaccurately classified a number of fish eating bird species as Most Sensitive (e.g., bald eagles classified as Most Sensitive), due largely to an inadequate review of the scientific literature.
- The toxicity reference values (TRVs) were selected in an arbitrary fashion and did not utilize the full spectrum of known toxicity (e.g. NOAELs and LOAELs), which resulted in unsupported overestimates of risk.
- The screening level estimates in the GES ERA assume that predatory mammals consume fish that are far too large; for example, at the extreme, GES assumes that mammals (e.g. mink) weighing one and a half pounds consume fish (carp) weighing as much as 16 pounds.
- Published data on local wildlife recovery that conflict with the conclusions of this screening level ERA, were ignored and a weight-of-evidence proofing of the HQ/HI values was not done.
  - The GES report fails to cite or reference other local studies concerning bald eagle populations, walleye reproduction, trends in fish data, fishing reports, etc.
  - The GES ERA also fails to note the long-term decline of PCDDs/PCDFs in Tittabawassee River fish tissue.
- The GES ERA creates an unproven (i.e. not validated by field studies) approach of estimating “Sediment Threshold Concentrations” (STC) that are unsubstantiated by any reference that supports this approach.
  - These STCs are then used to make asserts that broad, undesirable levels of risk to predatory birds and mammals exists along the entire length of the Tittabawassee River from Midland to the confluence, when in fact these values are based on biased sediment sampling conducted by MDEQ that provide limited characterization of the river and floodplain.

- This simplistic and unproven approach is incapable of mapping the areas in the floodplain that may, or may not, be impacting populations of birds and mammals.

- Published data on predatory birds, that conflict with the conclusions of the GES ERA, were ignored.
  - The Report fails to cite or reference other studies with bald eagles, walleye reproduction, trends in fish data, fishing reports, etc.
  - The GES ERA also fails to note the long-term decline of PCDDs/PCDFs in Tittabawassee River fish tissue.
  - GES's assertions on population effects are really unnecessary, and inappropriate in a Tier 1 Screening Level Risk Assessment Effort and inconsistent with other actual population studies and observations of the presence of the species.
- Toxicity reference values (TRVs) for some bird species were erroneously referenced in the GES ERA; for example, the GES Report estimated TCDD risk to bluebirds, when the supporting literature reference did not include bluebirds and other bird species that were studied were unaffected (see page 22 of this Response).

3. Dow Will Conduct A Definitive ERA as Part of the Remedial Investigation and Follow EPA Guidelines.

- Dow is required to conduct an ERA by Condition XI.B.3(b)(v) of its Hazardous Waste Site Operating License that was issued to Dow on June 12, 2003.
- For that ERA, Dow intends to follow the EPA Guidelines for ecological risk assessment.
- In addition, the ERA to be conducted by Dow will consider the guidance recently published by the National Academy of Sciences (2001) and the recent draft *Framework for Application of the Toxicity Equivalence Methodology for Polychlorinated Dioxin, Furans, and Biphenyls in Ecological Risk Assessment* (EPA, 2003).

DOW RESPONSE TO THE GES REPORT

The GES Report's Executive Summary

- The GES Report does not make a novel finding by reporting PCDD/PCDF in fish from the Tittabawassee River, given that a State of Michigan fish consumption advisory exists based only in part on PCDD/PCDF levels for several years. This is not unique, as the State of Michigan also has fish advisories for PCDD/PCDF in the watersheds of Lake Erie (Lake Erie, Detroit River), Lake Huron (Lake Huron, Tittabawassee River, Saginaw Bay, Cass River, Saginaw River), Lake Michigan (Lake Michigan, Green Bay, Torch Lake), and Lake Superior. The measured PCDD/PCDF levels in fish from the Tittabwassee River and other watersheds of the Great Lakes have been shown to be decreasing overall, as shown in Figures 1 (Tittabawassee River) and 2 (Lake Ontario). The GES Report provides no reference or perspective on these facts.
- The GES Report concludes that the concentrations of PCDD/PCDF in four of the five species of fish collected pose a reproduction risk to fish eating birds and mammals. These conclusions should be put in context with EPA's risk assessment guidelines regarding interpretation of hazard quotients or indices. As such, it would be more appropriate to

state that the PCDD/PCDF levels may pose a sufficient risk such that a definitive ERA should be conducted, which Dow will conduct as required under its License. A definitive ERA will more accurately reflect the actual conditions and thus, provide a more meaningful basis for making risk management decisions.

- Conclusions in the GES Report Executive Summary rest upon a number of elements (discussed in detail in this Response, below) that are not consistent with EPA Guidelines for anything other than a screening level ERA and even then, there are errors that should be corrected.
- The assumptions inherent in the GES ERA regarding piscivorous birds and mammals are best viewed as being screening-level in nature, due to the exaggerated exposure scenarios, inadequate analysis of available TRV values, and examination of only NOAEL values in assessing TRVs. Collectively, these factors lead to a significant overestimate of risk to piscivorous animals regarding PCDDs/PCDFs in the Tittabawassee River. The extent of the errors and exaggerated assumptions make it difficult to properly assess the relative risk of PCDD/PCDF congeners.
- There were technical problems with the limited bird egg data collected as part of the GES ERA and while the GES Report indicates that the egg data supports a conclusion of risk, in the end the GES Report does not rely upon the collected egg data, but instead calculates modeled (estimated) egg concentrations that are much higher than the actual results, which calls into question whether there is any significance at all in the limited egg collection data (since it was not used). The definitive ERA will collect many more eggs and take more work actually measurements of reproductive success and population data for increased confidence in remedial decision making.
- For the reasons summarized above and discussed in detail below, the GES Report's calculation of sediment threshold concentrations are inappropriate for a screening level ERA and the numbers calculated are an artifact of assumptions and errors in the GES Report. The exaggeration of risk represented by these calculations is demonstrated, in part, by the incredibly low concentration levels presented as being protective for mink and river otter, which are actually above the background soil level for PCDD/PCDF on a TEQ basis in 13 counties in Michigan as reported by MDEQ's Michigan Soil Background Dioxin Data Map prepared by the Hazardous Waste Division. The GES Report itself acknowledged that the MDEQ Tittabawassee River sediment sampling "*focused on depositional areas and avoided erosional areas*" which may have caused "*low risk*" areas to be undetected on the Tittabawassee River (GES Report p. 39). This biased, non-randomized sediment sampling, combined with the GES ERA exaggerated exposure, toxicity, and risk assessment HQ values, produces sediment corrective action levels that are highly speculative in nature. Given the unrealistic assumptions in the GES ERA and the nature of the incomplete, non-randomized MDEQ sediment characterization data on the Tittabawassee River, any conclusions reached regarding risk estimates and sediment cleanup levels are exaggerated and not based on the best available science.

1. INTRODUCTION (P. 7)

No technical comments were identified for this section of the GES report.

2. ERA – OBJECTIVES AND PROCESS (P. 7)

<u>Dow response</u>: The GES ERA does not conform to accepted practice in the area of risk assessment, as defined by the EPA and as GES purports to follow.

An ecological risk assessment (ERA) is an iterative process that evaluates the probability that adverse ecological effects are occurring or may occur as a result of exposure to one or more stressors, which may include chemicals. As such, ERAs generally consist of a number of tiers or levels, which increase in confidence or certainty with increased study and information. Under EPA Guidelines (EPA, 1998), a Tier I risk assessment is a simple worst-case estimation, which is conducted with limited data and assumes values for parameters that are scarce or lacking. For a Tier I or screening level ecological risk assessment, it is important to minimize the chances of concluding that there is no risk when in fact a risk exists and therefore, by its very nature and design, exaggerates risks. This technique assures that the probability of a false negative or finding of no risk is very low. Thus, use of minimal exposure and toxicity information is acceptable, because of the bias for overestimating risk. This ensures that sites that might pose an ecological risk are studied further. If a Tier I effort suggests a potential hazard, then a definitive risk assessment, which is a more detailed study of exposure and effects needs to be performed, including specifically designed toxicity tests or field evaluations.

A definitive assessment is necessary to define effective corrective goals and specific types of remedial action. As noted in EPA Guidelines (EPA, 1997):

> *[T]he SMDP (scientific/management decision point) made at the end of the screening-level risk calculation will not set a preliminary cleanup goal. Screening ecotoxicity values are derived to avoid underestimating risk. <u>Requiring a cleanup based solely on those values would not be technically defensible</u>* (emphasis added).

EPA practice[4], based on years of experience with remedial action decision-making, has been to employ prudent realism rather than excessive conservatism when an ERA may ultimately impact possible remedial actions and wildlife populations.

The GES ERA does not appear to follow this basic premise of EPA risk characterization policy, unless it is interpreted strictly as a screening-level ecological risk assessment. The small data set, estimated values, and inadequate toxicity assessment in the GES ERA indicate that this study can be considered, at best, a Tier I assessment of piscivorous wildlife on the Tittabawassee River. The GES ERA was based on a simple model for only one exposure pathway, exaggerated assimilation of consumed prey, did not account for dietary inputs of body weights of piscivorous species, used only no observed adverse effect levels (NOAELs) as the toxicity reference values (TRVs) in the calculation of hazard quotients (HQs), and did not consider weight-of-evidence. In addition, in most cases for avian species, the NOAELs were calculated, rather than measured, as they were extrapolated by use of 10x safety factors from LOAEL (at best) or presumed "field effect concentrations" (FECs). The LOAELs and/or FECs were presented in the GES ERA, but they were not used to calculate a range of HQ values or discussed in any uncertainty analysis regarding the HQs. Finally, site-specific data were ignored and reliance on an inadequate literature search resulted in erroneous conclusions about some piscivorous species.

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[4] EPA policy on this point was articulated by former Administrator Browner in her cover letter on EPA Guidance for Risk Characterization (EPA, 1995): "*While I believe that the American public expects us to err on the side of protection in the face of scientific uncertainty, I do not want our assessments to be unrealistically conservative. We can not lead the fight for environmental protection into the next century unless we use common sense in all we do.*"

A definitive ERA should be based on a wide variety of techniques for measuring and characterizing ecological risks at sites, such as described in guidelines for ERA from EPA (EPA, 1997 and 1998) and as recommended by the National Academy of Sciences (National Research Council, 2001); which Dow intends to consider when performing its definitive ERA as required under its License. These include measurements, not estimates, of the following:

- The abundance, diversity, and other characteristics of exposed invertebrate, fish, and wildlife communities, and
- The reproductive success in fish, birds, and mammals.

A more thorough evaluation of multiple lines of evidence would provide a defensible basis for further decision making or discussion. Additional lines of evidence would include many more site-specific measurements, such as a quantitative survey of local fish, prey, and wildlife populations and evaluation of site-specific dietary exposures, evaluation of tissue residue concentrations in receptors of concern, and measurements of reproductive success for certain receptors of concern (see Table 1). The use of Monte Carlo and other probabilistic approaches would allow characterization of the probability or likelihood that adverse effects might occur in wildlife as a result of exposure to PCDD/PCDFs in the Tittabawassee River watershed. The GES ERA does not meet these objectives and as currently written can, at best, serve the purpose of a Tier I, screening-level assessment, and cannot be used to support development of corrective action goals or other remedial action decisions.

2.1 The U.S. EPA Ecological Risk Assessment Framework (p. 8)

<u>Dow response</u>: Dow agrees that the EPA ERA Guidelines are the appropriate foundation of a technically defensible ERA. However, Dow disagrees with the premise that the GES ERA followed these Guidelines to the extent GES claims. By comparing the GES ERA with the EPA Guidelines, the GES ERA is at best a screening level ERA. For a variety of reasons discussed below in greater detail, it falls well short of being an ERA that can be used for remedial decision making.

Specifically, the GES ERA, as compared with the EPA Guidelines, is limited to a screening-level ERA in the following ways:

- The GES ERA is fundamentally based on limited data set (85 Tittabawassee River fish).
- The GES ERA has only one modeled (estimated) exposure pathway, that being estimated or exaggerated risk to fish-eating wildlife species.
- The GES ERA provides no direct site-specific data (measured values) for those wildlife species that were reported to be at risk.
- Site-specific information on Tittabawassee River walleye PCDD/PCDF levels were not used in the GES Report.
- Site-specific information addressing multiple lines of evidence was not considered in the GES Report.
- The risk levels asserted in the GES Report are inconsistent with the presence of breeding populations of picivorous birds and mammals species which are readily observed in the Tittabawassee River area.

- The approach used in the GES Report to calculate the hazard index (HI) or hazard quotient (HQ) was appropriate under EPA Guidelines only for a screening-level ERA and not for a definitive ERA.
  - This is due in part to the Toxicity Reference Values (TRVs) being based only on "No Observable Adverse Effect Level" (NOAEL) concentrations, rather than on both NOAELs and "Lowest Observable Adverse Effect Level" (LOAEL) concentrations, as recommended in EPA Guidelines.
  - The sole use of NOAELs is most appropriate for Tier I or screening-level assessments.
  - As noted in EPA guidance (EPA, 1997): "*screening ecotoxicity values should represent a NOAEL for long-term (chronic) exposures to a contaminant… A NOAEL is more appropriate than a LOAEL to use as a screening ecotoxicity value to ensure that risk is not underestimated*".
  - In most cases the GES ERA (particularly in avian species) the NOAELs were estimated using 10x safety factors from LOAELs (some of which were arbitrarily determined), rather than using statistically pertinent NOAEL values, when known.
  - Given that the assumptions in a screening-level ERA are intentionally exaggerated to ensure that risk is not underestimated, chemicals/locations that have HQ values greater than 1.0 merely indicates the need for further evaluation in a definitive ERA, which Dow intends to conduct as required under its license.
- EPA Guidelines recommend against the use of acute gavage studies instead of chronic feeding studies and reliance on secondary literature and sources which the GES ERA used to support some of its conclusions.
- The GES ERA also relies upon and cites studies that were outdated and does not reference or rely upon more recent and relevant publications on pertinent issues. This inadequate literature search resulted in erroneous conclusions about some piscivorous species. For example, the GES ERA indicates that bald eagles should be considered a "*most sensitive*" piscivorous avian species (i.e., egg NOAEL of 5-50 pg-TEQ/g), while recent research has indicated that NOAEL values are at least 100 pg/g and the LOAEL is 303 pg/g; both endpoints were based on enzyme induction, which is an adaptive change and not an actual adverse effect to the organism (i.e., very conservative endpoints). Elliott and Harris (2001/2002) noted the following:

  > *In summary, the results of this study are consistent with the emerging data from both field and laboratory studies which indicate that predatory birds are not particularly sensitive to some of the effects of TCDD. Assessments based on field studies on eagles (Elliott et al., 1996) and ospreys (Woodford et al., 1998) and the comparative egg injection work with kestrels, indicate that raptors are rather insensitive to some of the toxic and biochemical effects of TCDD and PCBs. Elliot et al., (1996) suggested a no-effect level (based on hepatic CYP1A in hatchlings) of 100 pg/g TEQs and a lowest effect level of 303 pg/g.*

Thus, a wealth of available data on bald eagles indicates that this species does not belong in a "*most sensitive*" species classification for PCDD/PCDFs, but rather has NOAEL and LOAEL values >100 pg/g and a "least sensitive" classification using the GES system is more appropriate, based on the published scientific data.

2.2 Uncertainty (p. 11)

<u>Dow response</u>: The GES ERA does not fully describe the uncertainties in its analysis and the impacts of these uncertainties on the conclusions of the ERA.

The failure to adequately address uncertainties underscores the nature of the GES ERA as a screening-level analysis. In general, the GES ERA failed to adequately discuss the ramifications of the substantial uncertainties that were inherent in its analysis and selection of TRVs for the avian and mammals species. There was limited discussion of the uncertainty associated with the fish to bird egg BMFs, limited focus on receptor groups, incomplete nature and extent characterization of the sediments, incomplete characterization of dietary composition of ecological receptors, a lack of data for concentrations of PCDD/PCDFs in those dietary items, and incomplete, non-randomized MDEQ sediment characterization data on the Tittabawassee River used in sediment characterization. Taken together, the magnitude of uncertainty from these areas significantly overestimates risk to piscivorous wildlife in the GES ERA.

3. PROBLEM FORMULATION (p. 11)

3.1 The Assessment Area (p. 11)

<u>Dow response</u>: The GES ERA's summary of the assessment area contains a number of statements that either need correction or references supporting their citation in the GES ERA.

The discussion in the GES ERA regarding the relationship between the river depth and the flood frequency would appear to go well beyond the available data and likely the expertise of GES.

In the paragraph at the top of page 13 there are statements about the Tittabawassee River and its floodplain that are not supported by any references and in fact are not accurate. For example, there is no support for the statement that "[i]*n wet years, the majority of the floodplain may flood up to a depth of several feet* (emphasis added)". Neither the term "majority" nor "floodplain" is defined for the river in terms of time frequency or depth, nor is it apparent from the lack of detail that GES is knowledgeable about flood dynamics on the River and the associated water flow issues. The accuracy of the statement regarding frequency and flood depth is not supported by any references to standard U.S. Geological Survey (USGS) data flow data on the Tittabawassee River. The GES ERA does not present available probability data from the USGS concerning discharge flows for ranked flood events, i.e., 5-year flood discharge, 10-year flood discharge, etc. It does not appear that GES is aware that USGS flood probability data for U.S. rivers report only discharge flow rates and that any conclusions regarding depth or extent of flooding for certain probabilities would require a hydrodynamic model for the Tittabawassee River. Dow is not aware that such a model exists, so it is unlikely that firm conclusions regarding the depth and frequency of flooding extent for the River can be responsibly decided.

Importantly, the GES ERA reports the presence of a variety of fish and bird species observed in 2002 and 2003 and the fact that some of the bird species (great blue heron, osprey, belted kingfisher, mergansers, and bald eagle) are known to breed along the Tittabawassee River (see p.

13). The reference to these observed populations is not used in the GES ERA in its assessment or in proofing of its contention of population-level harm to piscivorous wildlife. Omission of such multiple lines of evidence is consistent with a screening-level risk assessment process.

The GES Report purports to address the Saginaw River and Saginaw Bay, yet does not contain any discussion of the existence of PCBs and other confounding materials. No data is reported to have been collected as part of the GES ERA on the Saginaw River and Saginaw Bay. None of the ERA calculations and risk assessment results pertain explicitly to the Saginaw River and Bay, so this section of the GES Report should be disregarded entirely.

3.2 Contaminants in the Assessment Area (p. 15)

No technical comments. This information appears to be directly taken from the MDEQ Phase II report.

3.2.1 Structure, Toxicity, and Environmental Behavior of PCDDs/PCDFs (p. 15)

Dow response: The GES ERA should correct certain conclusions drawn from literature reviews that are not in fact supported by the referenced literature.

The GES ERA uses a secondary reference (Eisler, 1986) to suggest that PCDDs/PCDFs may "*elicit mortality in some organisms at concentrations as low as a few pg/g in tissues*" (p. 16). A more careful examination of the scientific literature finds that while dietary PCDD/PCDF TEQ concentrations of ~12 pg/g (Tillitt et al., 1996) may be capable of producing chronic reproductive impairment in a laboratory setting with mink (likely due in part to confounding other contaminants in Saginaw Bay carp), the levels resulting from this exposure are not "*a few pg/g*". Rather the mink liver TEQ concentrations ranged from 300-820 pg/g. This overstatement should be corrected in the GES ERA.

3.3 Conceptual Model (p. 16)

The simple conceptual models used in the GES ERA are acceptable for screening-level risk assessment purposes, yet are not acceptable for a definitive ERA or for remedial decision making.

3.4 Assessment Endpoints (p. 17)

Dow response: The GES ERA concludes that the two endpoints for assessment were protection of piscivorous avian and mammalian embryos and reproductive capacity and yet the GES ERA did not involve the collection of site specific measured values for either of these assessment endpoints.

The GES ERA is not based on site-specific measured values regarding either piscivorous avian or mammalian embryos, as only duck and chicken eggs were collected and even these duck and chicken egg measured values are not used in the final risk calculations. There were no site-specific measured values collected in any mammals. Population data on the presence of avian or mammalian species in the Tittabawassee River/watershed was not used in the risk analysis. Again, this approach would be adequate for a screening-level ERA, but not for a definitive risk assessment or for drawing definitive conclusions regarding risk to picivorous (fish eating) wildlife along the Tittabawassee River.

4. ANALYSIS (p. 21)

4.1 Avian Piscivores (p. 21)[5]

<u>Dow response</u>: The TRVs used to characterize risk of TCDD TEQs to piscivorous birds in the GES ERA are inappropriate and exaggerate risk. Some of the TRVs assigned to piscivorous receptors of concern are based on outdated scientific information.

The specific problems in the GES ERA analysis of TRVs for piscivorous birds are:

1. The TRV process used in the GES ERA likely do not meet standards of general scientific and statistical practice,
2. An inadequate literature review produced erroneous TRV classification of piscivorous receptors of concern, and
3. The basis of the specific TRVs that were selected was not described adequately in the ERA.

These problems are more fully described as follows:

1. <u>The TRV Process Used in GES ERA Likely Does Not Meet Standards of General Scientific and Statistical Practice</u>.

The approach of using a safety factors (i.e., 10x) to derive LOAEL/NOAEL concentrations and the use of only NOAEL data in the risk calculations is consistent with a screening-level ERA, not a definitive ERA.

EPA Guidelines (EPA, 1997; EPA, 1998) state that **<u>both</u>** a no observed adverse effect level (NOAEL) and a lowest observed adverse effect level (LOAEL) should be identified so that risk managers can clearly evaluate and provide context to the range of risk potentials that are presented. In the GES ERA, there were three different TRV classifications that were presented for birds but these TRVs were strictly based on NOAELs only and excluded consideration of LOAELs from the actual risk characterization analysis. More importantly, the decision to frequently interpret LOAEL values as a factor of 10 lower than the reported "field effect concentrations" (FECs) appeared arbitrary in nature, as the FECs themselves were often statistically defined by the authors of a given study as statistically significant LOAEL values ($P<0.05$). Similarly, the NOAEL values were extrapolated by the use of 10x safety factors and were not based on a statistically pertinent interpretation ($P<0.05$) of the data. The GES ERA's use of such revised, scaled NOAEL and LOAEL values is contrary to ordinary scientific and statistical understanding of the terms. Both NOAEL and LOAEL values are understood as typical endpoints in chronic exposure studies derived statistically by the hypothesis-testing approach, generally with an alpha requirement of $P<0.05$ (Rand, 1995). This is generally done statistically using a one-way analysis of variance (ANOVA), followed by a Dunnett's test for a pair-wise comparison of the endpoint means back to the control data. If the FECs have met the statistical requirement as LOAELs in the reported studies ($P<0.05$ for an appropriate effect endpoint), those values should be reported as LOAELs, not an arbitrarily scaled 10x lower concentration.

[5] The GES ERA does not define the acronym "PCH", which was used in this section of the document.

Similarly, for mink and otter, only one TRV was applied in ERA calculations and that value was based on a NOAEL only. For both birds and mammals, the basis of the TRVs appears to be the NOAELs, which are most appropriate, when used alone, for Tier I or screening-level assessments. As noted in EPA guidance (EPA, 1997): "*screening ecotoxicity values should represent a NOAEL for long-term (chronic) exposures to a contaminant… A NOAEL is more appropriate than a LOAEL to use as an screening ecotoxicity value to ensure that risk is not underestimated*".

2. An Inadequate Literature Review Produced Erroneous TRV Classification of Piscivorous Receptors of Concern.

The GES ERA did not attempt to statistically reanalyze the reported effect data ($P<0.05$), but applied 10x safety factors to derive both LOAEL and NOAEL that are not based on standard statistical practices for such measures. This approach is consistent with a screening-level ecological risk assessment, not a definitive ERA.

The derivation of TRVs for specific bird species was often arbitrary, not scientifically credible, and/or incomplete in the GES ERA. It is recommended that reasonable and well-justified TRVs be identified for specific bird species on both a NOAEL and LOAEL basis. As noted previously, the decision to interpret some LOAEL values as a factor of 10 lower than the reported "field effect concentrations (FECs)" is not consistent with the scientific and statistical understanding of LOAEL terminology. Similarly, some NOAEL values were extrapolated by the use of 10x safety factors and were not based on a statistically pertinent interpretation ($P<0.05$) of the data. There were numerous cases when reported NOAEL values were 10x or higher than the GES ERA-extrapolated NOAEL concentrations.

Dow's observations on the technical interpretation of the supporting avian toxicity studies cited in the GES ERA (Table 4-1, pp. 23-24) are as follows:

Wood duck

The GES ERA applies an excessive safety factor to derive a NOAEL that is lower than stated in the original report.

- In their work on wood ducks, White and Seginak (1994) noted in discussion of their field data that "*no reduction in nest success was detected until egg TEQs exceeded 20 ppt*"; the authors observed in their Abstract *that "the threshold range of toxicity where reduced productivity was evident in wood ducks was >20-50 ppt*". Despite this fact, the GES ERA LOAEL for wood ducks (p. 23, Table 4-1) is listed as "20-50 pg/g", not ">20-50 pg/g". The data presented by White and Seginak (1994) support the use of 20 pg/g as a field-derived no-effect level, but a calculated NOAEL of 2-5 ppt based on a 10x-safety factor was presented for wood ducks in the GES ERA. In this instance, the field derived no-effect level is approximately 4-10 times greater than the ERA-extrapolated value.

Great blue heron

The GES ERA used extrapolated (calculated) values rather than available (published) measured values to estimate Toxicity Reference Values (TRVs) for great blue heron.

Hart et al. (1991) found "*little or no difference*" in fertility and hatchability of great blue herons at contaminated (egg levels 250-550 pg/g) versus clean sites and the GES ERA TRVs are extrapolation from secondary effects (body weight, organ weight, tibia length, etc.) and other non-reproductive endpoints. The work of Henshel et al. (1995) on brain asymmetry has not been reproduced by other scientists and many believe this endpoint is not likely a useful avian biomarker for TEQ effects.

- In their work on brain asymmetry with great blue herons, Henshel et al. (1995) did not propose either a statistically supported LOAEL or NOAEL value ($P<0.05$). Examination of the data does not reveal a dose-related pattern of TCDD exposure and heron brain asymmetry and the authors did not report an FEC value or range in this work. The effects of co-contaminants (other than AhR active compounds) were not examined in this study. It appears that the TCDD TEQs reported in Henshel et al. (1995) are calculated from Safe 1990 TEFs, however, congener specific data are not presented. Both the NOAEL and LOAEL reported in the MDEQ GES ERA are extrapolated, non-statistically based values from a FEC and should be noted as such in ERA Table 4-1.

The LOAEL estimate of Henshel et al. (1995) is based on brain asymmetry and not on a reproductive endpoint. Subsequent publications investigating avian brain asymmetry have suggested that this response is dependent on physical factors, not TEQ concentrations. For example, Custer et al. (2001) states, "*A significant negative relationship between embryo brain asymmetry and the size of the egg suggested that physical constraint might be an important factor influencing the response of this bioindicator*." Lipsitz et al. (1997) evaluated the impact of PCB congeners on brain asymmetry with domestic chickens and found no statistically significant differences between exposed and non-injected controls ($P<0.05$). The authors suggested that asymmetry may be normal in avian brains "*resulting from both intrinsic structural asymmetry and extrinsic molding forces acting on the head during development*". In summary, the basis of the GES ERA estimating NOAEL/LOAEL values from this work despite the absence of a dose/response relationship appears not supportable. A thorough review of the subsequent scientific literature reveals that the brain asymmetry endpoint is not likely a useful avian biomarker for TEQ effects.

- The work of Hart et al. (1991) examined the impact of TEQs on great blue heron development. The data indicate "*little or no difference in the ability of fertile great blue heron eggs from either clean and dioxin-contaminated colonies to hatch successfully*"; the field dose levels of the sites studied ranged from ~245 to 560 pg/g (ww). The avian FEC reported in the GES ERA is based on alterations in body weight, tibia length, and organ weight, not heron great blue heron reproduction. Only five AhR-active compounds were considered in the chemical analysis and other possible chemicals of concern, such as DDE and other organochlorines, were not considered in regards to the observed toxicity. The FEC concentration was based on the TEQ concentration at the most contaminated site and the GES ERA LOAEL was then estimated by dividing this FEC value by 10. Therefore, Table 4-1 of the GES ERA should indicate (with an asterisk) that an uncertainty factor was used to calculate the LOAEL for Hart et al. (1991). The ERA's NOAEL TEQ value was calculated by dividing the ERA LOAEL by 10 and by using TEQ residues at the reference site, approximately 15-20 pg/g.

<u>Forster's tern</u>

The GES ERA arbitrarily applied safety factors to field effect concentrations (FECs) to generate NOAELs and LOAELs that result in inaccurate and over estimates of risk.

The GES ERA interpretation is of an effect concentration based on wasting syndrome in nestling birds and the NOAEL and LOAEL were estimated using 10x safety factors and were not statistically determined ($P<0.05$).

- Harris et al. (1993) collected eggs from Green Bay, Lake Michigan (10 eggs from 10 nests) for chemical analyses on PCBs (#77, 105, 126, and 169). Dioxins and furans were measured but not used in this analysis since comparisons were done with a 1983 data set (Kubiak et al., 1989) for PCB congeners. Endpoints measured were length of incubation period, percent hatch, and weight at hatching. Harris et al. (1993) cites a NOAEL of 7 μg/g PCB (endpoint = hatching success) and a NOAEL for Forster's tern was found to be 84 μg/kg/day based on a regression incorporating data from multiple years (embryotoxicity in 1983 and wasting syndrome in 1988). However, other chemicals of concern besides the aforementioned PCBs were not considered in regards to the observed toxicity. It appears that GES ERA reported a FEC based on observed wasting syndrome in nestling birds and that NOAEL and LOAEL reported in the GES ERA were estimated using 10x safety factors and were not statistically determined ($P<0.05$).

<u>Common tern</u>

GES inappropriately uses acute toxicity values from the literature to derive chronic NOAELs and LOAELs, which is inconsistent with appropriate scientific procedure as it leads to errors in interpretation. This practice is discouraged under EPA Guidelines, even for screening level risk assessments.

The GES ERA erroneously reported that PCB 77 levels were present and derived NOAEL and LOAEL values from acute LD50 data, not chronic information.

- Hoffman et al. (1995) collected eggs from a "clean location" with total PCB concentration = 1.5 mg/kg or less (no congener data supplied). Exposure began 4 days post-hatch and an LD50 for PCB 126 was determined to be 104 ng/g or 10,400 pg-TEQ/g. The GES ERA NOAEL and LOAEL values were derived from the LD50 data for PCB 126 and were not based on chronic developmental or reproductive endpoints. The GES ERA erroneously reports that the Hoffman et al. (1995) data included residues of PCB 77. There were no data for PCB 77 for the common tern, therefore reference to PCB 77 should be removed from Table 4-1 in the GES ERA. Guidance from the EPA (EPA, 1997) also discourages the use of acute gavage studies to derive effect concentrations for ERAs, even in screening-level ecological risk assessments, as chronic studies are preferred, along with more realistic modes of uptake:

  > *"To develop a chronic NOAEL for a screening ecotoxicity value from the existing literature, the following data hierarchy minimizes extrapolations and uncertainties in the value:*

- *A NOAEL is preferred to a LOAEL, which is preferred to an LC50 or an EC50.*
- *Long-term (chronic) studies are preferred to medium-term (subchronic) studies, which are preferred to short-term acute studies.*
- *If exposure at this site is by ingestion, dietary studies are preferred to gavage studies, which are preferred to non-ingestion routes of exposure."*

This EPA risk assessment data hierarchy indicates that data such as that developed for TCDD TEQ and the common tern are only appropriate for use in a screening-level ecological risk assessment, at best.

<u>**Cormorant**</u>

GES misrepresented published reports on cormorants and made other errors that resulted in an inaccurate representation of the available information. The GES ERA interpreted a chicken study as a cormorant experiment, based NOAEL/LOAEL extrapolations on acute LD50 data, and inappropriately compared H4IIE-based TEQs to chemically based (i.e. GC/MS) TEQs.

- Powell et al. (1997) injected chicken eggs with various concentrations of a cormorant egg extract (PCB 126 made up 70% of the toxicity). Endpoints measured in the chickens were mortality, developmental abnormalities, body weight, and organ weight. However, as cormorant egg extracts were used in chicken toxicity tests, this is actually a toxicity test for chickens, not cormorants, and should not be represented as such in Table 4-1. It is unknown how GES calculated the reported TEQ concentrations in the ERA.
- In another study with cormorants, Powell et al. (1998) injected eggs either with concentrations of PCB 126 or 2,3,7,8-TCDD in order to calculate an avian-based TEF for PCB 126. The GES ERA reported NOAEL and LOAEL values based on an acute LD50 for 2,3,7,8-TCDD (4,000 pg/g). Therefore the NOAEL and LOAEL values were based on acute mortality data and not chronic reproductive or developmental data. Powell et al. (1998) reports effects of 2,3,7,8-TCDD in regards to alterations in bursa and spleen weight at 11,700 pg TCDD/g egg, however this value was not used by GES to estimate either NOAEL or LOAEL values. It is suggested that the GES ERA examine other publications on cormorants, such as the work by Sanderson and Bellward (1995), who report developmental abnormalities in cormorant eggs exposed to 3,000 pg/g TCDD and noted an increase in total hepatic P450 content in cormorant eggs exposed to 10,000 pg/g TCDD.
- Tillitt al. (1992) collected cormorant eggs from the Great Lakes region and a reference site. Cormorant colonies were monitored for hatching success and eggs were extracted for PCBs. The PCB-containing extracts were tested on the H4IIE rat hepatoma cell line and TCDD TEQs were calculated and correlated with hatching success. The ERA's LOAEL was based on egg mortality data, not developmental or reproductive data. No dioxin or furan congener-specific data were reported in this paper, so WHO TEFs could not be used to calculate TCDD TEQs in this case. A significant issue is that the H4IIE-based TCDD TEQs reported in Tillitt et al. (1992) are not comparable with TCDD TEQs calculated from congener specific residue data (multiplied by WHO TEFs), as performed in other parts of the GES ERA or when TEQ is calculated from GC/MS-measured

congener concentrations. The H4IIE TCDD TEQ concentrations reported by Tillitt et al. (1992) should not be used in the GES risk assessment process, as there is no data or knowledge of other egg extract components and competing materials in the H4IIE bioassay. In addition, the H4IIE-based TEQs are not directly comparable with TEQs derived from WHO TEFs.

Pheasant

The GES ERA makes a number of errors in referencing the available information on pheasants and uses acute studies to estimate chronic effect concentrations (NOAEL/LOAEL), a practice discouraged by EPA, even for screening level risk assessments.

The Nosek et al (1993) study results were misrepresented in that effects were not seen at a dose level (100 pg/g) that GES cited as a level resulting in effects. The 1992 Nosek et al report is not applicable or is an incorrect citation, as it is a pharmacokenetic study which GES uses to support Toxicity Reference Values (TRVs). In the Brunstrom and Reutergardh (1986) report, GES mistakenly reports PCB 77 results as results for PCB 126 and also arbitrarily estimates an acute toxicity (i.e. LD 50) value as the mid-point between two dose groups, even though no effects were reported in the lower dose.

- Nosek et al. (1993) injected concentrations of TCDD into pheasant eggs on day 0 of embryonic development and endpoints were examined in day-old hatchlings and 28-day old chicks. The endpoints included EROD activity, mortality, body growth, organ weight, edema, histology of various organs, and carcass morphometrics. The MDEQ estimated LOAEL is based on embryo mortality and/or EROD activity. The NOAEL was calculated from the LOAEL by applying a safety factor of 10. Significant effects in embryo mortality and EROD activity were found at the 1,000 pg TCDD/g level, but not at the 100 pg/g level, so it is unclear as to why the LOAEL was reported in the ERA as '100-1000 pg/g' or '<1000 pg/g'. It is more accurate to report the LOAEL as 1,000 pg/g. At the 100 pg/g level there were no adverse effects observed on either growth or developmental endpoints (i.e., a no-effect level).

- Nosek et al. (1992) examined the elimination and partitioning of TCDD in pheasant hens, chicks, and eggs. However, it is not clear where effects on pheasants were studied in this paper, as it appears to be only a study on the distribution of TCDD in eggs (from the mother hen). It is possible that a different Nosek et al. (1992) paper should have been referenced here in the GES ERA (Nosek JA, Craven SR, Sullivan JR, Hurley SS 1992). Toxicity and reproductive effects of 2,3,7,8TCDD in ring-necked pheasant hens (J. Toxicol. Env. Health 35: 187-198). In this latter study, hen pheasants were injected with various doses of TCDD and mortality, wasting syndrome, and decreased egg production were observed; a LOAEL of 10 ng/g (10,000 pg/g) was reported to affect body weight, egg production, and mortality. The GES ERA reports a TCDD TEQ LD98 value of 3,300 pg/g and uses a safety factor of 10 to extrapolate NOAEL and LOAEL estimates from this reported LD98 value. It is unclear from the Nosek et al. (1992) publications how this LD98 value was calculated, and it seems incongruous that the same safety factors are applied to both LD50 and LD98 data to extrapolate NOAEL and LOAEL values. The LOAEL of 10 ng/g (10,000 pg/g) reported by Nosek et al. (1992), based on mortality and reproductive data, would be a more accurate effect concentration to reference in this case.

This experimental value is approximately 30x higher than the extrapolated LOAEL value of 33 presented in the GES ERA.

- Brunstrom and Reutergardh (1986) studied pheasant, mallard, and black-headed gull eggs that were injected with PCB 77 on embryonic day 4. Hatching rate was observed in the birds. At 100,000 pg/g (5,000 pg/g TCDD TEQ) PCB 77 did not cause embryo mortality above levels observed in the control. At 1,000,000 pg/g (50,000 pg/g TCDD TEQ) PCB 77 cause 100% mortality in pheasants. However, there were no data for PCB 126. The GES ERA erred in mistakenly treating PCB 77 data as PCB 126 data. Therefore the GES TEQ calculation was based on PCB 126 (WHO TEF = 0.1), not PCB 77 (WHO TEF = 0.05). In addition, GES estimated and reported the LD50 as a range with the upward bound number being the halfway point between the two reported data points in Brunstrom and Reutergarth (1986). This datum is highly uncertain given the study design. There were no adverse effects noted on hatchability in the lowest dose tested (5000 pg/g TEQ, when the GES ERA calculation is corrected for the WHO TEF value for PCB 77).

Turkey

GES misinterpreted published data on turkeys and made other errors that resulted in an inaccurate representation of the available information.

The GES ERA mistakenly reports PCB 77 results as results for PCB 126, and uses acute studies to estimate chronic effect concentrations (NOAEL/LOAEL), a practice discouraged by EPA, even for screening level risk assessments. It is also unclear on how the acute value for turkeys was derived, as no significant differences in embryo mortality were noted at the 40,000 pg/g dose level.

- Brunstrom and Lund (1988) injected PCB 77 into chickens and turkeys and relative toxicity was examined. The reported LD60 for PCB 77 in turkeys was reported to be 1,000,000 pg/g PCB 77 and exposure to 40,000 pg/g PCB 77 is reported to result in no significant differences in turkey embryo mortality. Again, the GES ERA misinterprets the report, as there were no data reported for PCB 126, as indicated in GES ERA Table 4-1; the GES ERA is mistakenly treating PCB 77 data as PCB 126 data. Therefore, the GES TEQ calculation was based on PCB 126 (WHO TEF=0.1), not PCB 77 (WHO TEF = 0.05). It is unclear how a TEQ concentration of 40,000 pg/g was derived by GES for the LD50.

Bobwhite

The GES ERA cited the incorrect primary reference for published bobwhite LD50 results and used secondary literature, which is strongly discouraged by EPA risk assessment guidance (EPA, 1997).

- The cited reference of Hoffman et al. (1996) is not the primary source for the reported PCB 126 LD50 value with this avian species. The primary source is Hoffmann et al. (1995), which is not a peer-reviewed journal source, but an abstract from a presentation. The use of secondary literature is strongly discouraged by EPA risk assessment guidance (EPA, 1997).

<u>Mallard; Herring gull; Domestic goose</u>

The GES ERA mistaken treated PCB 77 data as PCB 126 data, which results in a two-fold error in estimating risk, and extrapolated effect information from a study that reported no effects (of PCB 77) on embryonic mortality in mallards, herring gulls, or geese.

- Brunstrom (1988) worked with PCB 77 that was injected into duck, herring gull, goose, and chicken eggs on embryonic day 5. No abnormalities in mallard, herring gull or goose were noted in response to PCB 77 exposure (dosed at 1,000,000 and 5,000,000 pg/g PCB 77). There are no data reported for PCB 126. Once again, the GES ERA mistaken treated PCB 77 data as PCB 126 data. Therefore, the GES TEQ calculation was likely based on PCB 126 (WHO TEF=0.1), not PCB 77 (WHO TEF = 0.05), a two-fold error. In addition, exposure to PCB 77 caused no effect on embryonic mortality. A PCB 77 LD50 value reported in the GES ERA is estimated as the mean of the two tested concentrations. This LD50 is not based on any data reported by the authors and should not be used in the ERA.

<u>Eastern bluebird</u>

GES uses a reference for the eastern bluebird in which this species of bird was not studied and no effects were reported on any bird species included in the study. It is unclear how this report has any utility in estimating TCDD risk to the eastern bluebird.

- Thiel et al. (1989) examined wild bird populations (robins, chickadees, nuthatches, and wrens) in pine plantations in Wisconsin that were previously fertilized with TCDD contaminated sludge. The study concluded that, "*birds breeding in treatment plots had nesting success equal or better than individuals of the same species nesting in control plots*". There were no concrete data presented on eastern bluebirds. In addition, there were no reported effects on birds from TCDD-contaminated sludge. It is not clear how a TCDD LOAEL value of 10,000 pg/g was selected based on this study.

<u>Black-headed gull</u>

The GES ERA makes a number of errors in referencing the available information on black-headed gulls and uses acute studies to estimate chronic effect concentrations (NOAEL/LOAEL), a practice discouraged by EPA, even for screening level risk assessments. It is also unclear how the LD50 value was estimated, since there were no effects at the dose reported in the GES ERA.

- The work of Brunstrom and Reutergardh (1986) studied pheasant, mallard, and black-headed gull eggs that were injected with PCB 77 on embryonic day 4 and hatching rate was observed in the birds. At 1,000,000 pg/g (50,000 pg/g TCDD TEQ) PCB 77 did not cause gull embryo mortality above levels observed in the control, i.e., 50,000 pg/g TEQ appears to be a field no-effect level. There were no data reported for PCB 126. The GES ERA again is mistakenly treating PCB 77 data as PCB 126 data. Therefore, the GES TEQ calculation may have been based on PCB 126 (WHO TEF=0.1), not PCB 77 (WHO TEF = 0.05). GES estimated the LD50 as a range with the upward bound number being half the value of the concentration used in the study. This data point is highly uncertain given that it is based on no effect data. The GES ERA NOAEL and LOAEL values are extrapolated from this highly uncertain endpoint and the reported 50,000 pg/g TEQ value that did not cause gull embryo mortality above levels observed in the control was ignored.

Rock dove (domestic pigeon)

The GES ERA makes a number of errors in referencing the available information on rock doves and uses acute studies to estimate chronic effect concentrations (NOAEL/LOAEL), a practice discouraged by EPA, even for screening level risk assessments.

- TCDD was injected into pigeon eggs (air cell) on embryonic day 3.5 (1 ng/g) or embryonic day 14 (3 ng/g) by Janz and Bellward (1996); TCDD was also injected into great blue heron and chicken eggs. EROD activity, percent hatch, liver to body weight ratio, body and skeletal growth, and thyroid hormone concentrations were examined. Effects were seen on all parameters except thyroid hormone levels. The GES ERA selected 3,000 pg/g TCDD as the LOAEL value, however, growth and development of pigeons injected 3.5 days after fertilization were affected at 1,000 pg/g TCDD, so it is unclear why 3,000 pg/g TCDD was selected here as the LOAEL. In the discussion, a value of 2,000-3,000 pg/g TCDD (pg/g) was suggested as an LD50 for pigeons. ERA Table 4-1 should be revised to address this fact.

3. <u>The Basis of the Specific TRVs That Were Selected Was Not Described Adequately in the ERA</u>.

As noted in the GES ERA, there are substantially different species sensitivities among different bird species to PCDD/PCDFs. However, the GES ERA is in error when it cites TRVs for bald eagles that were primarily based on older work based on data primarily taken from domestic chickens and wood ducks (Table 4-2). Chickens are known to be among the most sensitive bird species to the effects of dioxin-like chemicals. For example, in Table 4-2 (page 25 of the ERA), it is noted that a NOAEL TEQ level of 20 pg/g was used in previous bald eagle assessments, based on the reference of Kubiak and Best (1991); this is a secondary reference to a TRV, again not a practice supported by EPA risk assessment guidance. Kubiak and Best (1991) based their bald eagle TRV on a laboratory domestic chicken TRV of 20 pg/g by Verret (1976), as bald eagle TRVs had not been determined at that time. Similarly, the GES ERA (Table 4-2 page 25) cites a NOAEL of 7 pg/g for bald eagles reported secondarily by both Giesy et al. (1994) and Bowerman et al. (1995). Again, these references are secondary sources and the TRV reference is for wood ducks, not bald eagles. A careful reading of these studies reveals that none of these publications based their TRVs on bald eagle toxicological studies since bald eagle TRV data were not available in at this time. In conclusion, of the three citations in Table 4-2 of the GES ERA reportedly supporting the inclusion of bald eagles as "most sensitive" species to PCDD/PCDFs (NOAEL >5 and <50 pg/g), none of the secondary publications were actually focused on bald eagle TRVs, but rather used chickens and wood ducks as surrogates.

It is inappropriate to apply chicken- or wood duck-based TRVs to piscivorous birds, such as the bald eagle, when more specific data do exist but were overlooked in the GES ERA literature review. Studies over the last several years indicate that piscivorous predatory birds, such as bald eagles, great blue herons, ospreys, and kingfishers are much more resistant to the effects of dioxins and related chemicals than gallinaceous birds such as chickens (Elliott et al., 1988; Elliott et al., 1989; Elliott et al.; 1996; Elliott et al.; 2001a; Elliott et al.; 2001b; Elliott and Harris, 2001/2002; Woodford et. al., 1998; Kennedy et. al., 2003; Henning and Brooks, 2003). For example, Elliott et al. (1996) studied bald eagle eggs collected from reference areas and from areas near pulp mills in British Columbia. The researchers suggested an eagle egg TEQ NOAEL of 100 pg/g and a LOAEL of 210 pg/g, both based on enzyme induction, which is an adaptive change and not an actual adverse effect to the organism (i.e., very conservative endpoints). In a recent re-analysis of their bald eagle egg data, Elliott and Harris (2001/2002) proposed an eagle egg TEQ LOAEL of 303 pg/g. The authors also noted the following:

> *In summary, the results of this study are consistent with the emerging data from both field and laboratory studies which indicate that predatory birds are not particularly sensitive to some of the effects of TCDD. Assessments based on field studies on eagles (Elliott et al., 1996) and ospreys (Woodford et al., 1998) and the comparative egg injection work with kestrels, indicate that raptors are rather insensitive to some of the toxic and biochemical effects of TCDD and PCBs. Elliot et al., (1996) suggested a no-effect level (based on hepatic CYP1A in hatchlings) of 100 pg/g TEQs and a lowest effect level of 303 pg/g.*

In summary, a wealth of available data on bald eagles indicate that this species does not belong in the erroneous GES-assigned classification of "*most sensitive*" species for PCDD/PCDFs, but rather has NOAEL and LOAEL values >100 pg/g and a "*least sensitive*" classification is more appropriate, based on the published scientific data.

Similarly, the suggestion in the GES ERA that great blue herons belong in the "most sensitive" avian species classification is not based on the most recent scientific data. The difficulties involving the GES ERA interpretation of the Hart et al. (1991) and Henshel et al. (1995) studies and their endpoints have been previously discussed. Publications dealing with great blue herons, PCDD/PCDFs, and reproductive success not cited in the ERA include work by Elliott et al. (1988), Elliott et al. (1989), Elliott et al. (2001a), and Harris et al. (2003). Data in Elliott et al. (1989) indicates that in great blue heron chicks, mean TCDD TEQs of 472 pg/g affected some biochemical and morphological variables but did not reduce survival of embryos and no effects of any type were noted at average egg TEQ concentrations of 91 pg/g (range = 39 to 145 pg/g). In Elliott et al. (2001a), the authors noted "*As previously reported for bald eagles (Elliott et al., 1996), great blue herons seem to be less sensitive to PCDDs and PCDFs than laboratory avian species*".

The GES ERA's avian TRV literature review on PCDD/PCDFs did not discuss work on an important piscivorous raptor species, the osprey. In a study of Wisconsin osprey, Woodford et al. (1998) concluded that the TCDD TEQ NOAEL for embryo survival was equal to or greater than 136 pg/g. Elliott et al. (2001b) reported that TCDD TEQ egg concentrations of 77 to 134 pg/g had no significant effect on hatching success of osprey chicks in the Pacific Northwest.

In summary, the GES ERA's categorization of bald eagles and great blue heron as "*most sensitive*" piscivorous species (NOAEL of 5-50 pg/g TEQ) for the Tittabawassee River is not technically defensible, as it is not supported by the scientific data. Based on a more complete review of the recent scientific literature, these piscivorous species and the osprey would be more appropriately classified as "*least sensitive*" for the GES ERA (NOAEL >100 pg/g). Finally, the use of wood duck egg data has questionable relevance to an assessment focused on potential risk to piscivorous birds since the diet of this species is characterized as "*seeds, acorns, berries, grain, aquatic and terrestrial insects, other invertebrates*" (Ehrlich et al., 1988).

Diet – Egg Biomagnification Factors (p. 25)

Dow response: Modeled concentrations of TEQs in eggs of piscivorous birds are dramatically overestimated, leading to an exaggeration of exposure and risk.

Relative to birds, the exposure model is overly simplistic and too generalized to be of use for estimating exposure to a specific avian species. In addition, the species and size classes of fish that were used in the model were generally inappropriate for piscivorous birds and this level of uncertainty is not reflected in the GES ERA. The GES ERA also failed to consider realistically the influence of migratory and dietary behavior in their assessment of exposure of piscivorous avian populations. Lastly, the GES ERA failed to effectively utilize the small amount of Tittabawassee River avian field data available, as the data did not support the simplistic fish/egg BMF model proposed in the ERA.

Specifically, the GES ERA conceptual model is flawed in its widespread use of biomagnification factors (BMFs) for predicting TEQs concentrations in piscivorous bird eggs, which are then used to calculate hazard quotients (HQs) for specific avian species. One of the key, cited publications in the GES ERA for application of the BMF model was a study by Braune and Norstrom (1989) in which BMFs for 2,3,7,8-TCDD and other PCDD/PCDF congeners were proposed based on herring gull and alewife (prey fish) levels from Lake Ontario. However, this study is inappropriate for deriving BMFs because the herring gull levels were collected from eastern Lake Ontario while the alewives were collected from western Lake Ontario. Other studies (not referenced in the ERA) have documented that tissue concentrations of dioxin-like compounds for fish and colonial fish-eating water birds can vary considerably among different locations on Lake Ontario (Borgmann and Whittle, 1992; Suns et al., 1993; Weseloh et al., 1995; Pekarik et al., 1998a; Pekarik et al., 1998b). In addition, field studies are difficult to interpret because the food items that comprise the birds' diet are not strictly known and the concentrations of TEQs in all dietary items are not known. For example, Environment Canada indicates that the diet composition of herring gulls in Lake Ontario consist of ~25% alewife, 30% smelt, 15% other fish, and 25% birds and other wildlife species (http://www.on.ec.gc.ca/wildlife/factsheets/fs_herring_gulls-e.html). Similarly, Fox et al. (1990) report the diet of herring gulls in the Great Lakes to consist of 39-92% fish, 5-42% insects, 0.5-21% garbage, and assorted birds, amphibians, worms, and crayfish. The residues of PCDD/PCDFs in other pertinent prey species was not considered or evaluated by Braune and Norstrom (1989) in their determination of BMF values for various PCDD/PCDF congeners.

Similar arguments as to the difficulty of TEQ BMF field interpretation and dietary/food web composition issues may be applied to the work of Jones et al. (1994) and Kubiak and Best (1991), cited in the GES ERA. In addition, biomagnification of TEQs is not appropriate, since dioxin/furan congeners are differentially enriched or depleted in biological matrices, as noted in the GES ERA itself. These difficulties may help explain, in part, the very wide discrepancy reported by Kubiak and Best (1991) for their calculated BMF values for TEQ (19 and 98, respectively) at two separate locations in Lake Huron. In addition, site specific factors such as food webs, variable dietary inputs, and differing prey species likely also played a role. The resulting high level of variability of the BMF values again indicates their usefulness should be restricted to a screening-level ecological risk assessment.

Finally, the GES ERA employs a technically inappropriate method for calculating a fish/egg BMF for 2,3,4,7,8-PCDF. The cited work of Van den Berg et al. (1987) examining eel-to-cormorant liver accumulation of 2,3,4,7,8-PCDF was mathematically combined with work of

Braune and Norstrom (1989) on herring gull livers/egg ratios for the same chemical. This BMF application in the ERA therefore assumes that eel/bird liver accumulation pharmacokinetics in cormorants in the Netherlands (Van den Berg et al., 1987) may be successfully applied to herring gull liver-to-egg distribution for organisms in Lake Ontario (Braune and Norstrom, 1989). This assumption requires that accumulation kinetics would be similar not only between different bird species, but also different feeding patterns and food webs, different water bodies, and even different continents. Available data indicate that cormorants (*Phalacrocorax sp.*) feed almost exclusively on fish (Ehrlich et al., 1988), while the diet of omnivorous herring gulls (*Larus argentatus argentatus*) consists not only of fish but also birds, garbage, and other wildlife species (Fox et al., 1990). Thus, the resulting ERA-extrapolated fish/egg BMF value of 64.6 for 2,3,4,7,8-PCDF (ERA Table 4-3) is a highly uncertain estimate of a BMF, and best suited to use in a screening-level risk assessment scenario only. Given the relatively significant impact of the modeled ERA exposure values on the predicted risk assessment outcome in the GES ERA, it would have been more prudent to simply measure site-specific concentrations of TEQs in ecological receptors of concern, rather than depend on estimated or modeled concentrations.

Accordingly, EPA guidance (EPA, 1997) relates literature-based BMF values to screening-level exposure and risk calculation estimates:

> *Bioaccumulation values obtained from a literature search can be used to estimate contaminant accumulation and food-chain transfer…at the* <u>*screening stage*</u> (emphasis added). *many environmental factors influence the degree of bioaccumulation, sometimes by several orders of magnitude the most conservative bioaccumulation factor (BAF) reported in the literature should be used in the absence of site-specific information".*

As indicated by EPA guidance, the GES ERA for the Tittabawassee River would be best served by measurement of site-specific concentrations in specific species of interest in order to remove substantial uncertainty in the exposure assessment. A higher tier or definitive ERA would provide such information.

The GES ERA did not perform a thorough examination of the Tittabawassee River MDEQ egg field data presented in Table 4-5 (p. 27) of the ERA. While the GES ERA used the presented Tittabawassee River field data to support a fish/egg BMF value of 1.0 for the congener 2,3,7,8-TCDF, the ERA does not mention the other Tittabawassee River field fish/egg BMF values for 2,3,7,7-TCDD and 2,3,4,7,8-PeCDF. The Tittabawassee River data presented in Table 4-5 support field BMF values of approximately 0.1-0.2 for 2,3,7,8-TCDD and 0.3 for 2,3,4,7,8-PeCDF. These values are markedly lower than the literature values for 2,3,7,8-TCDD and 2,3,4,7,8-PeCDF used in the GES ERA of 29 and 10, respectively (Table 4-4, p. 27). Therefore, it appears that the GES ERA used fish/egg BMF values from other locations in the U.S. and overseas to support modeled egg TCDD/TCDF levels of PCDDs/PCDFs for piscivorous birds, while selectively ignoring Tittabawassee River site-specific BMF values that were approximately 30-150 times lower than the published values for other locations.

Lastly, the toxicity equivalence (TEQ) approach utilized in the GES ERA as a principal method for assessing the risks of PCDD/PCDFs to wildlife, converts concentrations of PCDD/PCDF congeners to 2,3,7,8-TCDD equivalent concentrations (TEQ). The toxicity equivalency factors (TEFs) used in the ERA, are, according to their developers, half order-of-magnitude approximations (Van den Berg et al., 1998). Half-order rounding can lead to as much as a doubling of the apparent risk attributable to a particular congener. As such, the technique is not developed to the point where it allows the quantitative prediction of wildlife effects that are characteristic of definitive risk assessments and thus contributes additional uncertainty to estimates of potential ecological risk.

Lack of Multiple Lines of Evidence

As discussed in EPA Guidelines (EPA, 1997; EPA, 1998), multiple lines of evidence are critical to developing a defensible ERA. However, the GES ERA presents virtually no lines of evidence other than the screening-level HQs, and fails to present a framework for resolving any conflicting lines of evidence. For example, as discussed above, the GES ERA applied biomagnification factors (BMFs) to concentrations of PCDDs and PCDFs in fish to extrapolate (model) theoretical egg TEQ concentrations in piscivorous birds. However, the GES ERA had measured egg TCDD/TCDF levels (from 9 eggs)[6] available from the Tittabawassee River watershed. The measured concentrations of dioxin/furan-based TEQs in these individual eggs were about 4 to 2,700 times lower than the concentrations predicted by the BMFs in the GES ERA. Yet the predicted egg TEQ results, not the actual, MDEQ-measured egg data, were used to calculate hazard quotients for piscivorous birds and mammals on the Tittabawassee River. Thus, the GES ERA significantly overestimated the potential for risk.

Using predicted overestimates in a screening-level assessment is acceptable in the absence of measured, site-specific concentrations. It is not appropriate, however, to ignore existing measured values and rely solely on predicted overestimates. Moreover, in a more definitive ERA, multiple lines of evidence on wildlife populations may be generated through quantitative field studies. The Clinch River ERA (Clinch River, 1999) provides an example of an ERA in which multiple lines of evidence were evaluated. In the Clinch River ERA, five lines of evidence were developed for fish and two for piscivorous wildlife (EPA report available at: http://yosemite.epa.gov/water/owrccatalog.nsf). Other examples in which multiple lines of evidence were utilized in an ERA include the Housatonic River (EPA report available at: http://www.epa.gov/ne/ge/), Clark Fork (EPA report available at: http://www.epa.gov/region8/superfund/sites/mt/millterap.html), and the Hudson River (EPA report available at: http://www.epa.gov/hudson/revisedbera-plates.pdf).

Errors and Omissions

There is a calculation error in the GES ERA in the concentrations of TEQ for fish collected in the Tittabawassee River in 2002 (page 29, Table 4-7). The total TEQ (based on avian TEF values) for bass is presented as 73 pg/g, whereas the actual calculated TEQ value should be 63 pg/g. This error should be corrected in the text.

Additionally, it is unclear why MDEQ-collected walleye TEQ residues (9 walleye caught and analyzed) from the Tittabawassee River were deliberately excluded from the GES ERA analysis, despite this fish being a key species for piscivorous birds and mammals on the Tittabawassee River watershed. The walleye whole fish TEQ concentration (based on avian TEFs) averaged approximately 45 pg/g and approximately 15 pg/g when based on mammalian TEFs.

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[6] The highest report PCDD/PCDF concentration in a wood duck egg was from an egg taken outside of the Tittabawassee River floodplain.

4.2 Mammalian Piscivores (P. 30)

<u>Toxicity Reference Values</u>

<u>Dow response:</u> In the GES ERA, the basis of the TRV of 1 ng TEQ/kg or 1 pg/g (ww in diet) for mink and otter is unclear, as the derivation of TRVs for mink was not discussed in sufficient detail in the GES ERA. In addition, a number of studies cited in the GES ERA were based on mink feeding studies using Saginaw Bay carp.

There are substantial problems with using the mink feeding studies using Saginaw Bay carp (i.e., Giesy et al., 1994; Heaton et al., 1995; Tillitt et al., 1996; Restum et al., 1998), as the basis for a TRV. These studies were designed to assess reproductive and developmental toxicity in mink exposed through the diet to carp from Saginaw Bay, MI. However, there is substantial uncertainty in developing TRVs from these studies because other contaminants were present in fish from the Saginaw Bay which may not be present at the same concentrations in dietary items of mink from the Tittabawassee River (Giesy et al., 1997). It is inappropriate, therefore, to strictly assign causality of mink reproductive toxicity from Saginaw Bay carp strictly to PCDD/PCDF TEQ contaminants, as other materials may have been present to influence the reproductive endpoints of interest in the studies. This may explain why the TRVs resulting from the Tillitt et al. (1996) study are among the most conservative values for TCDD TEQ.

Ideally, TRVs should be developed from studies that were conducted with minimal impact of co-contaminants. Other problems with these Saginaw Bay carp studies are that there were few dose levels tested, the NOAEL was the control feed and the LOAEL was defined as the lowest dose tested. Therefore, the identified NOAEL and LOAEL are artifacts of dosing and consequently, there is considerable uncertainty regarding the actual threshold for effects from these studies with the true threshold for effects being somewhere between the control dose and the lowest dose. The magnitude between these dose levels may be considerable in some cases.

The GES ERA's assessment of the study of Brunstrom et al. (2001) is not consistent with the authors' stated conclusions. The ERA notes (p. 30) the lowest level of contamination in the diet that resulted in reproductive impairment (reduced kit survival) was 22 pg/g TCDD TEQ (LOAEL). While this LOAEL is consistent with the conclusions of Brunstrom et al. (2001), the GES ERA then applies an uncertainty factor of 10 to derive a NOAEL of 2.2 pg/g TCDD TEQ in the diet. This NOAEL value is contrary to that stated by Brunstrom et al. (2001), who noted "*The TEQ exposure in that group was…3.2 pg/g-feed…., which represents no-observed adverse effect level in terms of reproductive effects in our study*". The MDEQ ERA therefore misstated the reproductive NOAEL of this publication through an extrapolation technique. This excessively conservative, calculated TRV for mink is again consistent with a screening-level ERA approach.

Secondary sources (Kannan et al., 2000, Giesy and Kannan, 1998, etc.) were also relied upon to develop the mink TRVs in the GES ERA. The use of secondary literature in ERAs is strongly discouraged by EPA guidance (EPA, 1997), which states that:

> *When reviewing the literature, one should be aware of the limitations of published information in characterizing actual or probable hazards at a specific site. EPA discourages reliance on secondary references because study details relevant for determining the applicability of findings to a given site usually are not reported in secondary sources. <u>Only primary literature that has been carefully reviewed by an ecotoxicologist should be used to support a decision</u>.* (emphasis added).

In summary, it is recommended that TRVs for mink be selected that are reasonable and well justified. One useful study is Bursian et al. (2003), a mink feeding study conducted for EPA (Bursian et al., 2003). In this study, Bursian et al. (2003) fed Housatonic River fish to mink in a chronic laboratory study and determined a NOAEL, threshold dose, and LOAEL of 16.1, 33.2, and 68.5 pg TEQ/g (ww in diet), respectively based on sensitive and ecologically-relevant reproductive and developmental endpoints. The TRVs from the Bursian et al. (2003) study are strengthened by the fact that the dose intervals were very close together (only 2-fold differences among dose levels) and there were five doses at which there were no observable adverse effects on reproduction (e.g., 1.1, 3.5, 5.7, 9.2, 16.1 pg TEQ/g). The TRV of 1 pg TEQ/g that was used in the GES ERA is included among the concentrations tested by Bursian et al., (2003), and there were no observable adverse effects on mink reproduction at this dose level.

The use of a single TRV based on measured and extrapolated NOAEL values, as was done in the GES ERA, is consistent with a screening-level approach to risk assessment, but not with a definitive ERA. If TRVs such as that of Bursian et al. (2003) were used, the predicted risks to mink and otter would be substantially less than currently estimated. Estimated risks would drop even further if the Tittabawassee River walleye TEQ data were included in the exposure analysis.

5. RISK CHARACTERIZATION (p. 32)

5.1 Avian Piscivores (p. 32)

<u>Dow response</u>: The GES ERA contains errors and significantly overestimated both exposure and toxicity to piscivorous birds of concern on the Tittabawassee River, resulting in a significant exaggeration of risk potential for PCDD/PCDFs with these species. Revised calculations and toxicity value indicate mean HI/HQ values of approximately 1.0 for receptors of concern (i.e., bald eagles and great blue herons). The revised screening-level HQs for these species of interest (birds with TRVs >100 pg/g) vary from 0.6 to 2.3, suggesting minimal or low risk of PCDD/PCDFs in fish for these important avian piscivore species on the Tittabawassee River.

There is an error in GES ERA Table 5-2 (p. 33). Clearly, the congener 2,3,7,8-TCDD could not contribute 148% of the TEQ via a carp-egg magnification. This value should be 14%.

As previously indicated, the oversight of the GES ERA with regard to erroneous NOAEL/LOAEL values for piscivorous receptors of concern (i.e., bald eagles and great blue herons) were perpetuated in this risk analysis section; such birds were erroneously identified as having "*most sensitive*" NOAEL TRVs of 5-50 pg/g (egg), based on research with wood ducks and chickens. Studies over the last several years indicate that many piscivorous predatory birds, such as bald eagles, great blue herons, ospreys, and kingfishers are much more resistant to the effects of dioxins and related chemicals than gallinaceous birds such as chickens (Elliott et al., 1988; Elliott et al., 1989; Elliott et al.; 1996; Elliott et al.; 2001a; Elliott et al.; 2001b; Elliott and Harris, 2001/2002; Woodford et. al., 1998; Kennedy et. al., 2003; Henning and Brooks, 2003). The GES ERA quotes a EPA (1993) document as supportive of fish TEQ concentrations of 60 pg/g or more as posing a "high risk" to sensitive bird species. However, this document was written and published several years prior to a majority of the recent research work on piscivorous predatory birds, such as bald eagles, great blue herons, ospreys, and kingfishers. A more recent literature search in the GES ERA would have revealed these species to be more resistant than assumed in the early 1990s, as the previous publications merely assumed these species to be as sensitive as gallinaceous, non-piscivorous species.

Despite the existence of Tittabawassee River field measured TEQ egg levels, the GES ERA used predicted egg TEQ results, not the actual, MDEQ-measured egg levels, to calculate hazard quotients for piscivorous birds on the Tittabawassee River. Using predicted values is acceptable in the absence of measured, site-specific concentrations, but ignoring measured values and relying on predicted overestimates is indicative of a screening-level ecological risk assessment. The GES ERA-estimated TEQ levels in piscivorous bird eggs were 4 to >2700 times greater than measured TEQ residues in eggs (N = 9) collected by MDEQ in 2002 at a reference site and the Shiawassee National Refuge. This is due in part to the GES ERA application of literature-based fish/egg modeled BMF values, which may not well represent the complex nature of PCDD/PCDF interaction between piscivorous birds and the aquatic environment on the Tittabawassee River. The GES ERA has presented data indicating that the literature-based BMFs vary from measured Tittabawassee River fish/egg BMFs by ~30-150 fold for some congeners.

This wide discrepancy between literature fish/egg BMF values for some PCDDs/PCDFs and Tittabawassee River field BMFs clearly underlines the screening-level nature of the GES ERA. However, the difference between field (Tittabawassee) and literature BMF values of 0.2 to 29 for 2,3,7,8-TCDD (a factor of ~150) and 0.3 to 10 for 2,3,4,7,8-PeCDF (factor of ~30) allows for calculation of revised, screening-level HQ values for the GES ERA categories of sensitivity for avian piscivores. If the Tittabawassee River field BMFs for 2,3,7,8-TCDD (BMF=0.2) and 2,3,4,7,8-PeCDF (BMF=0.3) are used along with the remaining, default BMF values in GES ERA Table 4-4 to calculate revised bird egg TEQ values, the resulting egg concentrations and HI/HQ values are presented in Text Table 1 (below):

Text Table 1. Estimated TEQ (pg/g, ww) in eggs of avian piscivores exposed to PCDD/PCDFs in fish from the Tittabawassee River, based on revised, field BMF values. Also presented are revised HI values, based on predicted, modeled egg TEQ.

	Carp	Catfish	Shad	Bass	Walleye	Mean
Egg TEQ (pg/g, ww)	232	87	225	58	57	132
HI (5 pg/g)	46	17	45	12	11	26
HI (50 pg/g)	4.6	1.7	4.5	1.2	1.1	2.6
HI (100 pg/g)*	2.3	0.9	2.3	0.6	0.6	1.3

* Data indicates that bald eagles, great blue herons, and osprey all have NOEL values ≥100 pg/g.

The "revised", calculated egg TEQ values presented in Text Table 1 indicate a mean egg residue of 132 pg/g, with a range of 57 to 232 pg/g. These revised predicted values are closer to the mean MDEQ-measured egg concentrations for Tittabawassee River wood ducks (154 pg/g) and hooded mergansers (288 pg/g) than the GES ERA-calculated egg TEQ values (mean = 1031 pg/g, range 333 to 2222 pg/g). As shown in Text Table 1, the revised, field-based BMF values had a significant impact on the screening-level HI values, with the revised HI values for avian piscivores with NOAEL TRVs of ≥100 pg/g (i.e., bald eagle, osprey, great blue heron) varying from 0.6 to 2.3, with a value of 1.3 for mean fish TEQ residues. These screening-level findings suggest minimal or low level risk from PCDD/PCDFs in fish for avian piscivores on the Tittabawassee River.

These revised screening-level HQ risk estimates of approximately 1.0-2.0 for avian piscivores with TRVs >100 pg/g are reinforced by weight-of-evidence from field observations on the Tittabawassee River and Saginaw Bay watershed. As noted in the GES ERA (p. 13), numerous avian piscivore species are resident and active on the Tittabawassee River. In addition, field data from the Michigan Office of the Great Lakes (2002) indicates that there is an increased rate of bald eagle reproduction and nesting success in the Saginaw Bay watershed, as demonstrated by an increasing temporal trend for bald eagle nests and young production for Saginaw Bay (see Figure 3). Bald eagles nesting along the Saginaw River have now exceeded the goal set for reproductive recovery of 1.0 young per occupied nest (Michigan Office of the Great Lakes, 2002; Bowerman, 2001). These data suggest improving wildlife populations for such avian piscivore receptors of concern on the Tittabawassee River and Saginaw Bay watershed area.

In summary, the GES ERA significantly overestimated both exposure and toxicity to piscivorous birds of concern on the Tittabawassee River, resulting in a significant exaggeration of risk potential for PCDD/PCDFs with these species. The ERA's categorization of bald eagles and great blue heron as "*most sensitive*" piscivorous species (i.e., NOAEL of 5-50 pg/g TEQ) for the Tittabawassee River is not technically defensible, as it is not supported by the available scientific data. The fish/egg BMF model is overly simplistic and literature BMF values were selected in the GES ERA over site-specific parameters. Use of the latter, "revised", site-specific BMF values produces egg concentrations closer to measured MDEQ 2002 data for Tittabawassee River birds than predicted by the GES ERA; the resultant HI values for avian receptors of concern (i.e., bald eagle, great blue heron – species with NOAEL TRVs of >100 pg/g) were approximately 1.0. These findings suggest minimal or low risk of PCDD/PCDFs in fish for these important avian piscivores on the Tittabawassee River. Weight-of-evidence from proximal field studies in the Saginaw Bay watershed reinforces these findings, as the data indicate an increasing rate of bald eagle reproduction and nesting success in the Saginaw Bay watershed (Figure 3) and eagles nesting along the Saginaw River are now exceeding 1.0 young per occupied nest (Michigan Office of the Great Lakes, 2002; Bowerman, 2001). The avian piscivore risk characterization in the GES ERA does fulfill EPA guidance for a Tier I or screening-level ERA but it does not establish a defensible basis for making remedial decisions. The results of the GES ERA, along with the "revised" ERA HQ values presented here, indicate the need to continue through the process of conducting a higher tier or definitive ERA as specified in the Hazardous Waste Management Facility Operating License, which was issued on June 12, 2003 by MDEQ to Dow.

5.2 Mammalian Piscivores (p. 33)

<u>Dow response</u>: The GES ERA mink feeding assumptions (e.g. up to 100% fish in diet and consumption of extremely large fish) with Tittabawassee River fish (excluding walleye) may be appropriate for a screening-level risk assessment, however, a more complete ERA would focus on the actual distribution of mink food consumption data.

The fish composition of the mink's diet was estimated at 54.6% by Sample and Suter (1999), based on a mean value of five studies. Application of the Bursian et al. (2003) TRV of 16 pg/g (mink reproduction NOAEL) to the fish TEQ data produced HI/HQ values of 0.3 to 3.2 with mean fish TEQ residues. These screening-level results can only be interpreted that further study of the piscivorous mammals on the Tittabawassee River is warranted (i.e., HQ may be less than 1 or slight greater than 1) via a definitive ERA.

Relative to mink, the assumption that these organisms eat 100% fish is not realistic and results in an overestimate of risk. Mink are opportunistic feeders and have a diverse diet, as documented in the EPA Exposure Factors Handbook (1993). Specifically, the work of Selander (1943) reported that Michigan mink may consume up to 43% of their diet as muskrat, 16% as rabbits, 18% as birds, 10% frogs, and less than 5% fish. Data from Michigan trout streams indicate that 52-56% of the mink's diet may be trout and 6-26% non-trout fish. The fish composition of the mink's diet was estimated at 54.6% by Sample and Suter (1999) in the Clinch River aquatic ERA, based on a mean value of five studies. Once again, these data indicate that while the GES aquatic ERA mink feeding assumptions (up to 100% fish in diet) on Tittabawassee River fish may be appropriate for a screening-level risk assessment, a more complete aquatic ERA would focus on the actual distribution of mink food consumption data available in the scientific literature. Table 5-4 of the ERA (p. 35) that described alternative proportions of fish in the mink diet is useful to illustrate the impact of this assumption but needs to be coupled with a more appropriate TRV (discussed later) as a basis for a more realistic distribution of potential risk.

In addition to mink dietary composition, the relative size of fish captured and analyzed by MDEQ on the Tittabawassee River in 2002 is also pertinent when discussing potential risk of PCDD/PCDFs for mink. Data on mink in Michigan, collected by Alexander (1977) and cited by Sample and Suter (1999) in the Clinch River GES ERA for piscivorous mammals, indicate that on average 72% of fish captured and consumed by mink are less than 10 cm in length and the remaining 28% are less than 20 cm in length. Examining the fish length data for Tittabawassee River fish collected by MDEQ in 2002 and used in the MDEQ GES ERA (see Text Table 2), all collected species averaged significantly greater than 10 cm in length and only one species, the shad, had an average length that was less than 20 cm.

Text Table 2. Average length of fish collected in 2002 by MDEQ on Tittabawassee River, compared to preferred fish size distribution for mink.

Mink: Prey fish size distribution[1]	Average (+ standard deviation) fish length (cm), as collected by MDEQ in 2003 on Tittabawassee River				
	Carp	Walleye	Catfish	Bass	Shad
0 - 10 cm = 72% 11 - 20 cm = 28%	56 + 9	49 + 2	44 + 9	35 + 2	16 + 6

[1] Alexander, GR. 1977. Food of vertebrate predators on trout waters in north central Michigan. Mich. Acad. 10: 181-195. Cited by Sample and Suter (1999) for Clinch River GES ERA.

This marked distinction in the average fish size used in the GES ERA versus actual prey fish size reported in the mink diet may significantly exaggerate the exposure portion of the GES ERA for mink. Concentrations of lipophilic chemicals have been observed to positively correlate with fish size and weight in the Great Lakes ecosystem (Manchester-Neesvig et al., 2001). Similar concentration/fish size data have been reported by MDEQ correlating fish length with TEQ carp concentrations (Michigan Fish Contaminant Monitoring Program, 2001 Annual report, available at http://www.deq.state.mi.us/documents/deq-swq-gleas-fcmpreport2001.pdf). These findings collectively suggest that the net result of the significantly larger Tittabawassee River fish sampled by MDEQ for use as biota inputs in the ERA may result in an overestimate of TEQ fish exposures for mink. It is recommended that additional site-specific data be collected and incorporated into the dietary model for mink/otter to improve the reality of the ERA and to reduce the uncertainties in estimating ERA exposures. The use of the current MDEQ fish residue data may only be appropriate for use in a screening-level ecological risk assessment.

The difficulties and lack of clarity associated with GES ERA-selected TRV of 1 pg/g (ww) for mink/otter has been extensively discussed. If TRVs such as that of Bursian et al. (2003) were used in the GES ERA, then the predicted risks to mink and otter would be substantially less than currently estimated, particularly if the MDEQ Tittabawassee River 2003 walleye fish TEQ data were also included in the total fish exposure analysis. As shown in Text Table 3, the mean fish TEQ concentration would be conservatively set at 51 pg/g if MDEQ walleye residue data were included in the overall analysis, and these residues are likely less for mink-consumed smaller fish, as noted in Text Table 2 (above). Application of the Bursian et al. (2003) mink TRV of 16 pg/g (reproduction NOAEL) to the fish TEQ data produced HQ values ranging from 0.1 (walleye and bass) to 8.0 (carp), with mean fish HQs of 0.3 to 3.2 (see Text Table 3). Of particular interest are the HQ values for mink diets of ~50% and below, as the reported range of mink diet as fish may be approximated at 5% to ~55% (Selander, 1943; Alexander, 1977; Sample and Suter, 1999). The resulting HQ values range from 0.3 (10% fish) to 1.6 (50% fish), based on mean fish residues, indicating a low or minimal risk to sensitive piscivorous mammals on the Tittabawassee River. These screening-level results merely indicate that further study of the piscivorous mammals on the Tittabawassee River is warranted (i.e., HQ may be less than 1 or slightly greater than 1).

Text Table 3. Fish TEQ concentrations for five fish species sampled on the Tittabawassee River by MDEQ (2002) and mink hazard quotient or index values, according to the percentage of fish consumed in their diet.

	Fish TEQ (based on mammalian TEFs) in pg/g (ww)					
	Carp	Catfish	Shad	Bass	Walleye	Mean
	128	50	44	18	15	51
%Fish	HI or HQ values, based on TRV of 16 pg/g (ww)*					
100	8.0	3.1	2.8	1.1	0.9	3.2
90	7.2	2.8	2.5	1.0	0.8	2.9
80	6.4	2.5	2.2	0.9	0.8	2.6
70	5.6	2.2	1.9	0.8	0.7	2.2
60	4.8	1.9	1.7	0.7	0.6	1.9
50	4.0	1.6	1.4	0.6	0.5	1.6
40	3.2	1.3	1.1	0.5	0.4	1.3
30	2.4	0.9	0.8	0.3	0.3	1.0
20	1.6	0.6	0.6	0.2	0.2	0.6
10	0.8	0.3	0.3	0.1	0.1	0.3

* NOAEL from Bursian et al. (2003) for mink

These screening-level findings may be proofed by a weight-of-evidence approach with local field population data. There are measurable, long-term reductions in loadings of chemical contaminants in the Great Lakes, as indicated by Figures 1 and 2 for walleye and lake trout with 2,3,7,8-TCDD. Such declines have likely aided in the recovery of populations of piscivorous birds (see Figure 3) and mammals in the Great Lakes. Data from the Michigan Office of the Great Lakes (2002) indicate fish-eating mammals (mink and otter) have shown trends of increasing populations. Mink populations in the Lake Huron watershed reportedly began recovery during in the 1980s. During the past five years, there have been increases in both the range and population of river otters within the Michigan portion of the Lake Huron watershed, including the Saginaw Bay (Michigan Office of the Great Lakes, 2002). For example, river otter are now routinely observed at the Nayanguing Point Wildlife Area and nearby Tobico Marsh (part of the Bay City Recreation Area). River otter have also colonized Crow Island State Game Area and the Shiawassee River State Game Area, and within the last three years have also been annually observed or trapped in Tuscola County (Michigan Office of the Great Lakes, 2002). These population trend data tend to support the weight-of-evidence that the screening-level HQ values presented in Text Table 3 for piscivorous mammals are reasonable for the Tittabawassee River watershed and that further evaluation is warranted via a definitive ERA.

In conclusion, the GES ERA significantly overestimates Tittabawassee River TEQ exposure to mink/otter from Tittabawassee River fish, both in terms of dietary composition and size of fish consumed, as the latter is correlated with TEQ fish body residues. Additionally, the GES ERA-selected TRV for mink/otter was taken from studies confounded by co-contamination with numerous Ah receptor active chemicals, and secondary sources were relied upon to support the selected mink/otter TRV. Finally, the use of a single TRV based on measured and extrapolated NOAEL values, as done in the GES ERA, is consistent with a screening-level approach to risk assessment, not a definitive ERA. Given the uncertainties noted in terms of both exposure and toxicity and the omission of site-specific data, the current mink/otter hazard quotients or indices noted in the GES ERA likely exaggerate risk by approximately 1-2 orders of magnitude (i.e., 10 to 100 times). A reanalysis of these fish TEQ data using a recent mink reproductive TRV from Bursian et al. (2003) reveals HQ values of 0.1 to 8, depending on the diet. These screening-level ERA results merely indicate the need to continue through the process of conducting a higher tier or definitive ERA.

5.3 Summary of Risk Characterization (p. 37)

Technical comments were covered in detail in previous sections 5.1 and 5.2.

6. RISK-BASED SEDIMENT CONCENTRATIONS (p. 38)

6.1 Sediment Threshold Concentrations of PCDDs/PCDFs (p. 38)

<u>Dow response</u>: A screening-level ERA is exaggerated by design and contains too much inherent uncertainty to be the basis for regulatory decisions such as attempting to propose sediment concentration levels.

The GES ERA calculated Sediment Threshold Calculations (STC) by taking a supposed “mean TCDD concentration of surface sediments” from the Tittabawassee River dividing that number by the Hazard Indexes (HIs) calculated by GES in Section 5 of the GES ERA. The STCs are then represented to be the ecologically “safe” sediment TEQ level in pg/g (dw). The GES-calculated STCs ranged from 9 pg/g (for river otters) to a high of 211 pg/g for the least sensitive bird eggs. However, the GES ERA does explain that “*using TEFs in this way does not imply any potential direct toxicity linkage between the sediments and the receptors, (since the risk to the receptors is expressed through food chain transfer of contaminants from sediments to exposed resources). It does provide useful accounting tool for identifying sediment TCDD-Q concentrations of concern*.” (p. 38).

However, the GES ERA modeling technique for extrapolating STCs from modeled hazard quotients for PCDDs/PCDFs in fish and piscivorous birds and mammals is fundamentally flawed due to the exaggerated exposure concentrations and the erroneous, unrealistic single value toxicity reference concentrations for both birds and mammals. The technique fails to consider dietary preferences for mink and field data supporting both avian and mammalian wildlife population increases for the Tittabawassee River watershed. These cumulative errors likely produce STC values that are exaggerated by of at least 10-100 fold.

6.2 Tittabawasse River (p. 39)

Dow response: The deliberately biased sampling, combined with the GES ERA exaggerated exposure, toxicity, and risk assessment HQ values, produced sediment cleanup levels that are highly speculative in nature.

The GES Report acknowledges that the MDEQ sediment sampling "*focused on depositional areas and avoided erosional areas*" which may have caused "*low risk*" areas to be undetected on the Tittabawassee River (GES Report 39). This biased, non-randomized MDEQ sediment sampling, combined with the GES ERA exaggerated exposure, toxicity, and risk assessment HQ values, produces sediment cleanup levels that are highly speculative in nature. It is also worth noting that the EPA STC values are from a March 1993 document, which was written before the most recent avian studies indicating that piscivorous predatory birds, such as bald eagles, great blue herons, ospreys, and kingfishers are much more resistant to the effects of dioxins and related chemicals than gallinaceous birds such as chickens (Elliott et al., 1988; Elliott et al., 1989; Elliott et al.; 1996; Elliott et al.; 2001a; Elliott et al.; 2001b; Elliott and Harris, 2001/2002; Woodford et. al., 1998; Kennedy et. al., 2003; Henning and Brooks, 2003). The EPA work also predated the most recent mink reproduction toxicity study with Ah receptor chemicals (Bursian et al., 2003). Given the overly conservative assumptions in the GES ERA and the nature of the incomplete, non-randomized MDEQ sediment characterization data on the Tittabawassee River, any conclusions reached regarding risk estimates and sediment cleanup levels are likely exaggerated and not based on the best, most recent science.

6.3 Saginaw River and Bay (p. 45)

Dow response: When considering weight of evidence on wildlife populations, results from the Saginaw River and Bay do not support the conclusions reached in the GES ERA for piscivorous (fish eating) birds and mammals.

Data from the State of Michigan Department of Environmental Quality and the Michigan Office of the Great Lakes (2002) indicate piscivorous birds and mammals have demonstrated an increasing population trend over the past few years in the Tittabawassee River and Saginaw Bay watersheds. Field studies in the Saginaw Bay watershed indicate an increasing rate of bald eagle reproduction and nesting success in the Saginaw Bay watershed, and mink populations in the Lake Huron watershed reportedly began recovery during in the 1980s. During the past five years, there have been increases in both the range and population of river otters within the Michigan portion of the Lake Huron watershed, including the Saginaw Bay. These population trend data collectively tend to support the weight-of-evidence that the screening-level HQ values presented in the GES ERA for piscivorous birds and mammals are exaggerated and that the impact of PCDDs/PCDFs on the Tittabawassee River watershed must be further evaluated via a definitive ERA.

7. UNCERTAINTIES (p. 47)

<u>Dow response</u>: In the GES ERA, it is acknowledged that there are uncertainties for certain assumptions. However, there is inadequate analysis of the potential magnitude of uncertainty and its impact on exposure and toxicity parameters and the risk potential. The current uncertainty analysis is acceptable, only if the GES ERA is properly interpreted as a screening-level risk assessment.

Each of these uncertainties has been addressed in other sections of this Response. Additional uncertainties that were not discussed in the GES ERA include the limited focus on only one exposure pathway, limited focus on receptor groups, incomplete nature and extent characterization, incomplete characterization of dietary composition of ecological receptors, a lack of data for concentrations of PCDD/PCDFs in those dietary items, failure to examine site-specific information, and incomplete, non-randomized MDEQ sediment characterization data on the Tittabawassee River. Taken together, the magnitude of uncertainty from these areas results in a significant overestimate of risk to piscivorous wildlife in the GES ERA. Attempts to revise HQ calculations based on these errors produced HQ values of 0.3 to 8 for mink/otter and 0.6 to 2.3 for piscivorous birds of interest, such as bald eagles, osprey, and great blue herons (TRVs >100 pg/g).

Without additional data, the only areas of uncertainty that can be effectively addressed include the selection of more appropriate and technically defensible TRVs for both avian and mammalian species. The other areas of uncertainty cannot effectively be addressed without collection of additional site-specific data, including evaluation of multiple lines of evidence. In other words, another iteration involving a higher tier or a more definitive ERA would be the next step based on EPA Guidelines.

The primary areas of uncertainty mentioned were:

7.1 Diets of Piscivorous birds and Mammals in the Assessment Area (p. 48)

The GES ERA does not attempt to mathematically express in its HQ analysis an understanding of the dietary variability with regard to fish size for the piscivorous birds and mammals in the Tittabawassee River area. Numerous authors have examined both the size and amount of fish prey consumed by piscivorous birds and mammals, yet no attempt was made in the GES ERA to correct for this variation, particularly with piscivorous birds. For GES ERA-identified avain piscivorous receptors of interest, such as the bald eagle and great blue heron, no attempt was made to modify HQ values by attenuating fish size, as only the 85 MDEQ-collected fish were used for the TEQ avian exposure analysis. As noted previously, the GES ERA was selective in its composition of the piscovore diet by excluding consideration of the collected walleye data without explanation and notwithstanding the comment that the avian and mammalian piscivores were opportunitistic in their prey selection.

The conduct of a definitive ERA, under the EPA Guidelines, as Dow will be conducting under the requirement of the License, will greatly reduce the uncertainties identified in the GES Report and in this Response.

7.2 Avian and Mammalian TRVs (p. 49)

- Avian TRVs:

  The GES ERA's misidentified numerous piscivorous birds as being "*most sensitive*" piscivorous species due to an incomplete review of the scientific data. Clearly, the ERA would benefit greatly from proper recognition of the correct TRV for important piscivorous species such as the bald eagle and great blue heron.

- Mammalian TRVs:

  The selected TRVs for mink and otter are incomplete and rely on NOAEL values only. Selection and use of only NOAEL TRVs in the GES ERA is consistent with a screening-level ERA, not a definitive ERA, according to USEPA guidelines. More recent mammalian TRVs are available and were not used in the GES ERA.

7.3 Fish – Bird Egg BMFs (p. 50)

The avian fish/egg BMF exposure model was overly simplistic and generated modeled concentrations of TEQs in eggs of piscivorous birds are dramatically overestimated, leading to an exaggeration of exposure and therefore risk. The GES ERA-calculated egg residues via this model were ~4 to 2,700 higher than measured egg residues available from the Tittabawassee River watershed. Despite this overestimation, the GES ERA used the predicted egg residues, not the actual, MDEQ-measured egg data, to calculate hazard quotients for piscivorous birds and mammals on the Tittabawassee River. Site-specific BMF values for some congeners helped to estimate egg TEQ levels that were closer to actual levels than predicted via the fish/egg BMF values from the literature.

7.4 Sediment Threshold Concentrations (p. 50)

The GES ERA modeling technique for extrapolating STCs from modeled hazard quotients for PCDDs/PCDFs in fish and piscivorous birds and mammals is fundamentally flawed due to the exaggerated exposure concentrations and the erroneous, unrealistic single value toxicity reference concentrations for both birds and mammals. The technique fails to consider dietary preferences for mink and field data supporting both avian and mammalian wildlife population increases for the Tittabawassee River watershed. These cumulative errors likely produce STC values that are exaggerated by of at least 10-100 fold.

7.5 Saginaw River and Bay (p. 51)

Data from the State of Michigan Department of Environmental Quality and the Michigan Office of the Great Lakes (2002) indicate piscivorous birds and mammals have demonstrated an increasing population trend over the past few years in the Tittabawassee River and Saginaw Bay watersheds. Field studies in the Saginaw Bay watershed indicate an increasing rate of bald eagle reproduction and nesting success in the Saginaw Bay watershed, and mink populations in the Lake Huron watershed reportedly began recovery during in the 1980s. During the past five years, there have been increases in both the range and population of river otters within the Michigan portion of the Lake Huron watershed, including the Saginaw Bay. These population trend data collectively tend to support the weight-of-evidence that the screening-level HQ values presented in the GES ERA for piscivorous birds and mammals are exaggerated and that the impact of PCDDs/PCDFs on the Tittabawassee River watershed must be further evaluated via a definitive ERA.

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Table 1. Examples of Data Collected for Ecological Risk Assessments at Large Sites

Location	**Study Area**	**Analytical Data Used in ERA**	**Source of Information**
Kalamazoo River Area of Concern, Michigan	80 Miles	> 400 fish > 200 mammal samples > 200 aquatic/benthic invertebrates > 190 bird samples > 100 terrestrial invertebrates > 40 plant samples	Information source: (MDEQ) Michigan Department of Environmental Quality and CDM. 2002. Final (Revised) Baseline Ecological Risk Assessment Allied Paper, Inc./Portage Creek/Kalamazoo River Superfund Site. Rep. January 2002; also data from studies conducted by Michigan State University
Fox River Area of Concern, Wisconsin	39 Miles	> 750 fish > 220 bird samples > 12 mammal samples 8 crayfish 31 aquatic/benthic invertebrates	Information source: http://www.dnr.state.wi.us/org/water/wm/lowerfox/background.html
Housatonic River, Massachusetts	11 Miles	> 1,400 fish > 75 mammal samples > 35 aquatic/benthic invertebrates > 420 bird samples > 55 amphibians > 100 terrestrial invertebrates > 60 crayfish	Information source: http://www.epa.gov/region01/ge/thesite/restofriver/reports/final_era/List_of_Volumes.pdf

Figure 1. Residues of 2,3,7,8-TCDD in Tittabawassee River walleye fillet, Dow dam, 1983-2002.

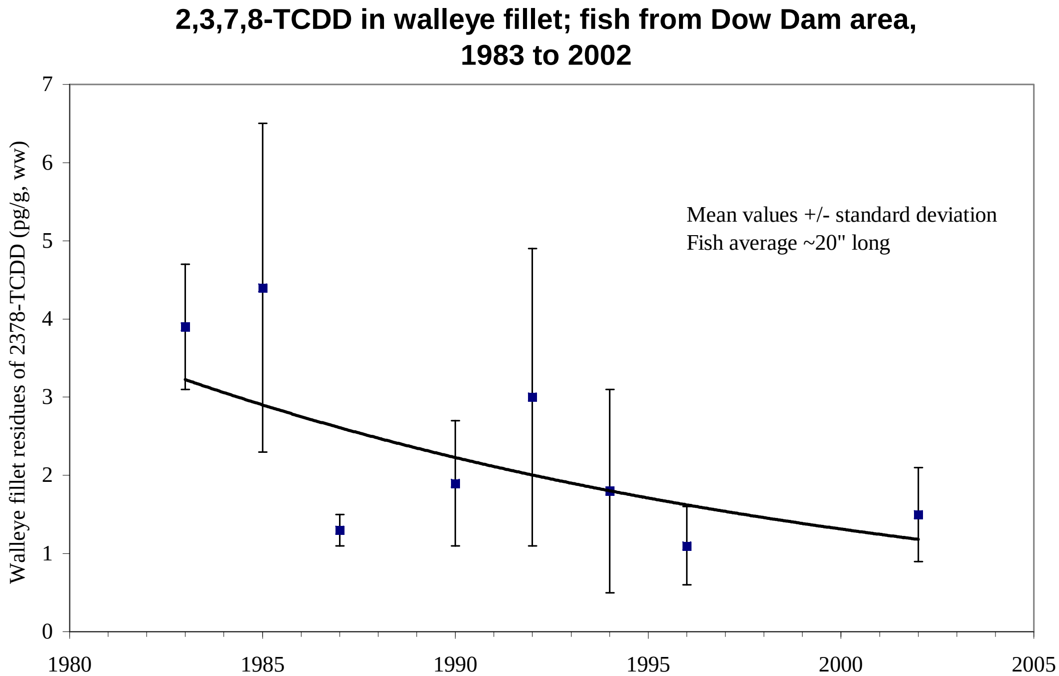


Figure 2. Residues of 2,3,7,8-TCDD in Lake Ontario lake trout, 1977-1993. Data from Huestis, SY, Servos, MR, Whittle, DM, Van Den Heuvel, M and Dixon, DG. 1997. Environ Toxicol Chem 16: 154-164.

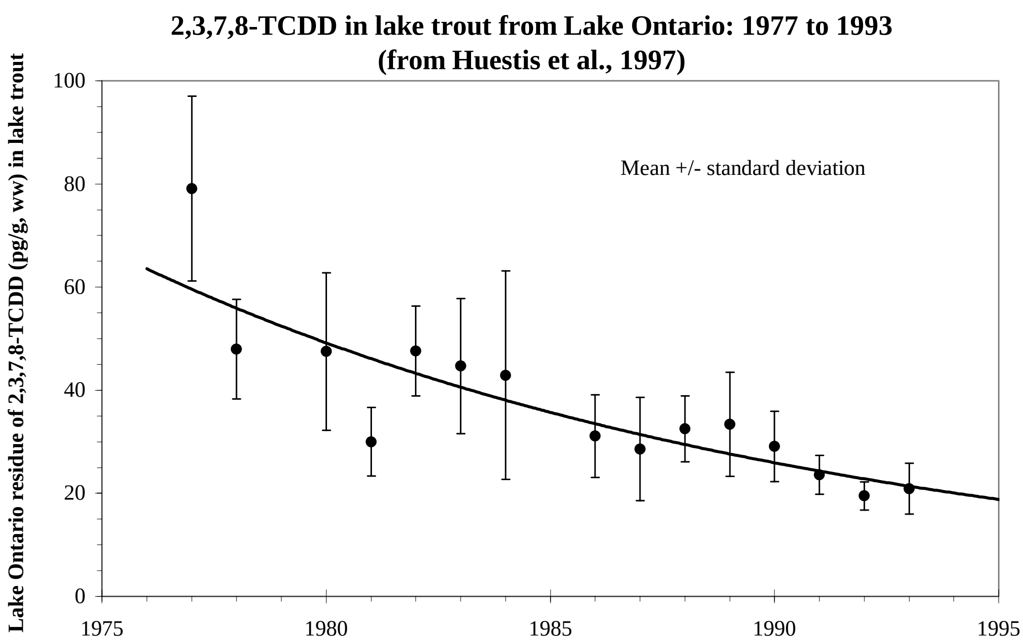
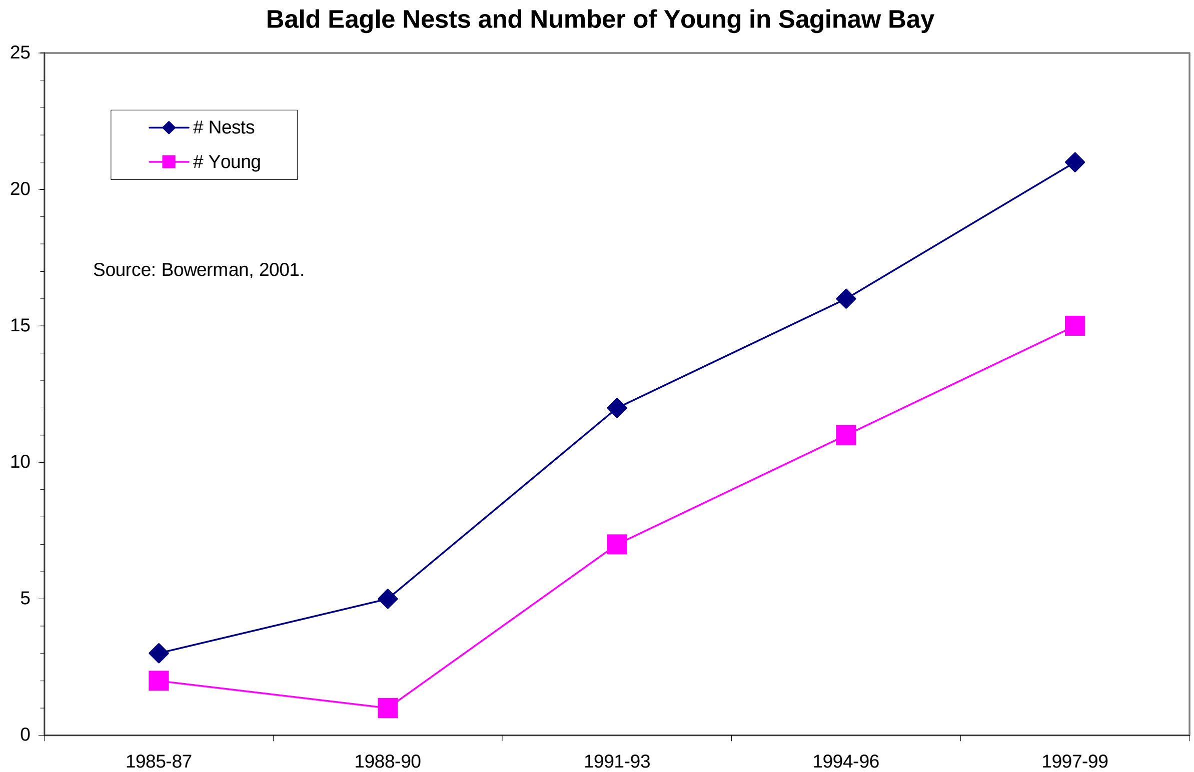


Figure 3. Temporal plot of number of bald eagle nests and number of young in Saginaw Bay, MI. Source: Bowerman, WH. 2001. Recovery of Bald Eagles in Lake Huron, 1991-2000. Clemson, SC: Department of Environmental Toxicology, Clemson University.



Definitions and Acronyms

BAF	Bioaccumulation Factor
BMF	Biomagnification Factor
CAP	Community Advisory Panel
Dow	The Dow Chemical Company
ERA	Ecological Risk Assessment
GES	Galbraith Environmental Sciences
HQ	Hazard Quotient: the ratio of estimated exposure to the toxicity reference value (i.e., the toxicity reference value is the dose of chemical assumed to be without deleterious effect for the receptor of concern, generally given in units of mg/kg-day, mg/kg, or pg/g). Hazard quotient = Hazard index (HI)
LOAEL	Lowest Observed Adverse Effect Level
MDEQ	Michigan Department of Environmental Quality
NOAEL	No Observed Adverse Effect Level
PCDDs	Polychlorinated Dibenzo-p-Dioxins
PCDFs	Polychlorinated Dibenzofurans
RI	Remedial Investigation
STC	Sediment Threshold Concentration
TRVs	Toxicity Reference Values
EPA	United States Environmental Protection Agency
WW	Wet Weight